THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—32ND YEAR. SYDNEY, SATURDAY, JUNE 23, 1945. No. 25.

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THE PATHOLOGICAL LESIONS IN 5,000 AUSTRALIAN AUTOPSIES.

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FROM 1920 to January, 1943, 5,000 autopsies have been performed at the Royal Adelaide Hospital and the results tabulated. The tabulation has been done entirely by myself. The majority of the autopsies have also been performed by me, although I have performed only about half of the last 2,000. I have, however, scrutinized the results, and when necessary have examined specimens. The tabulation has thus been the work of one person, and although my views have altered somewhat from time to time as further experience was gained, the general arrangement has been the same throughout. These 5,000 cases do not comprise all the post-mortem examinations performed over that period of twenty-four years. I was away ill for six months in the beginning of 1930 and was absent in Great Britain for another six months in the beginning of 1938. Also, a few autopsies have been so poorly recorded that I have omitted them from consideration.

The method of recording is as follows. At the end of each autopsy the important lesions present are set down in order of importance or in order of sequence, whichever seems better to suit the individual case. At the end of this summary appear such items of little significance as a Meckel's diverticulum or hydrocele. The post-mortem summary therefore indicates clearly what lesions were considered to be responsible for the death, and also shows all the associated lesions. The pathological conditions set down do not include trivialities, so that the summary is not overloaded. Each summary is then multityped and copies of it are pasted on cards under headings comprising the various pathological lesions present. For instance, the following, which appears under the heading of "coronary atheroma of cardiac importance" (*The Medical and Scientific Archives of the Royal Adelaide Hospital*, number 23, 1943, page 825) also appears under "calcified aortic cusps" (*loco citato*, page 804) and will in later issues be placed under "duodenal ulcer", "cerebral softening" and "melanosis of the colon". The entry is:

105/40. M.76. Chronic duodenal ulcer, melæna. Extensive softening in the floor and side of the left lateral ventricle. Aortic stenosis with calcified knobby cusps. Atheroma and calcification in the coronaries, especially near the commencement of the descending branch of the left. Some melanosis of colon.

The post-mortem results are tabulated in groups of a thousand each. The earlier thousands usually took about five years to perform, but for the later thousands only a little over three years was required. By tabulating the results in this way in thousands, one can see at a glance whether there has been any considerable increase or decrease in the incidence of any pathological lesion over the period of twenty-four years.

The results under the various pathological lesions have finally been rearranged in some order and then published regularly in *The Medical and Scientific Archives of the Royal Adelaide Hospital*, where the information is available for anyone who desires to know the incidence of various forms of disease in Australia. Copies of these archives have been forwarded as issued to the various public, university and medical libraries in Australia, as well as to medical libraries abroad.

With the completion of 5,000 autopsies it has seemed advisable to assess the results and see what information of value is forthcoming. In this initial paper it is proposed to give the broad results; it will be followed later by further contributions dealing with the details in connexion with the various systems of the body.

The question arises as to whether the incidence of disease here revealed can be taken as representing the incidence of disease in the adult inhabitants of South Australia as a whole. To a certain extent this seems to be the case, though there are unfortunately various circumstances which show that the persons on whom the post-

mortem examinations have been made have been to some extent selected. For instance, 3,302 of the 5,000 persons were males and 1,692 were females, the sex of the other six not having been entered. The number of deaths among women in the community must, of course, be more or less equal to that among men, and it would seem either that men were more likely to die in hospital than women or, what is perhaps a better explanation, that the relatives are more likely to grant permission for an autopsy on a man than on a woman. It is also probable that old men are more difficult to look after when serious illness overtakes them than are old women, and consequently are taken to hospital, whilst the old women may be nursed at home. In the early part of the series, infectious diseases, pulmonary tuberculosis and advanced cancer all had to be treated at the Adelaide Hospital; but during the last 3,000 and more autopsies these three types of cases have been catered for elsewhere, and the persons dying from these diseases in the Royal Adelaide Hospital have been mostly those who have been suddenly overtaken with some catastrophe or illness so acute as to require urgent hospital attention, or else the nature of the case was not recognized. Apart from this, the figures probably represent fairly closely the incidence of disease among the adults of the State. For instance, the finding of about 2% of hydatid cysts, usually old ones, at these post-mortem examinations probably means that about 2% of the adult inhabitants of South Australia are harbouring these cysts.

Permission is always asked before an autopsy is performed. An examination of the head is made whenever possible, though very often the autopsy is granted without permission to open the skull. In probably a little less than half of the cases the head was examined. In a few instances permission was granted merely to examine the brain, as, for example, in the presence of suspected cerebral tumours. Permission for an autopsy is frequently refused, so that those subjects on whom post-mortem examinations are carried out probably represent only about one-third of the total number who die in the Royal Adelaide Hospital. An effort is made in every case to secure permission for an autopsy, but, naturally, in those instances in which the disease is of particular interest, greater pressure may be exerted on the relatives than in simpler cases.

Sex and Age.

The attached figures (Table I), giving grand totals and, separately, those for males and females of the various age groups, show the number of autopsies carried out in the various periods of life. In the total figures it will be seen that 1,041 persons were in the age group sixty to sixty-nine years; this figure is followed closely by 948 in the age group fifty to fifty-nine years. Among males again the maximum of 747 was in the age group sixty to sixty-nine years, but in the case of women the highest number was in the age group fifty to fifty-nine years—namely, 316—294 being in the sixty to sixty-nine years class. There were necessarily very few autopsies on children aged under ten years, these cases being merely "emergencies", as most children would go to the Adelaide Children's Hospital. The oldest man was aged one hundred and two years, the next ninety-five years and the next again ninety years. The oldest woman was aged ninety years; the next two were aged respectively eighty-nine and eighty-eight years. The persons concerned were almost entirely of British ancestry, with some admixture of German. There were two Greeks, six Italians, two Maltese, one Negro, seven natives of India (Lascars and Bengalese), four Chinese, one half-bred Chinese-Japanese, one full-blooded Australian aboriginal, and twelve Australian aborigines with European blood (five males and seven females).

Diseases of the Vascular System.

Pericardial adhesions indicating some old pericarditis were found in 116 cases (2·3%). Pericarditis was present in 121 (2·4%). There were only two examples of tuberculous pericarditis.

Chronic rheumatic valvular defects occurred in 149 cases (3%), bacterial endocarditis in 112 (2·2%), acute rheumatic vegetations in 11, and other valvular lesions in 53 (1%). Syphilitic disease of the aorta had extended to the valve in 33 (0·66%).

Hypertrophied and dilated hearts, not due to valvular disease or renal disease, and presumably attributable to hyperpiesis, were responsible for heart failure in 196 cases (nearly 4%). In addition there were 72 cases in which hypertrophy of the heart was associated with hæmorrhage in the brain, and at the last 2,000 post-mortem examinations 27 cases were specially classified because the hypertrophied heart, presumably due to hyperpiesis, was associated with cardiac infarction. In addition there were 22 cases of hypertrophied heart with acute or subacute nephritis, and a number of cases in which some renal fibrosis was present. At the earlier post-mortem examinations, at a time when it was thought that these hypertrophied hearts were usually due to renal fibrosis, we often tried to convince ourselves that some slight renal fibrotic change was responsible for the high tension causing the hypertrophy. Consequently our figures were high under this category. For instance, 74 in the first 1,000 becomes reduced to 12 in each of the last two thousands; this figure

TABLE I.

Thousands.	Age Group (Years).										Age Doubtful.	Sex Doubtful.	Total.
	Under 10	10 to 19	20 to 29	30 to 39	40 to 49	50 to 59	60 to 69	70 to 79	80 to 89	90 and Over.			
						Both Sexes: Grand Totals.							
First	9	61	83	120	133	166	202	113	36	2	69	6	
Second	16	44	113	140	168	165	195	118	29	2	10	—	
Third	6	60	103	101	152	192	206	142	31	1	6	—	
Fourth	—	40	86	101	165	213	207	147	39	—	2	—	
Fifth	1	40	84	85	117	212	231	173	54	—	3	—	
Total ..	32	245	469	547	735	948	1,041	693	189	5	90	6	1,000
							Males.						
First	2	41	44	73	86	126	156	95	30	2	57		712
Second	8	29	59	78	121	106	140	104	19	2	9		675
Third	3	31	48	49	92	123	144	103	21	—	4		618
Fourth	—	26	40	50	90	136	145	114	28	—	2		631
Fifth	—	19	49	54	79	141	162	124	35	—	3 (old men)		666
Total ..	13	146	240	304	468	632	747	540	133	4	75		3,302
							Females.						
First	7	20	39	47	47	40	46	18	6	—	12		282
Second	8	15	54	62	47	59	55	14	10	—	1		325
Third	3	29	55	52	60	69	62	39	10	1	2		382
Fourth	—	14	46	51	75	77	62	33	11	—	—		369
Fifth	1	21	35	31	38	71	69	49	19	—	—		334
Total ..	19	99	229	243	267	316	294	153	56	1	15		1,692

probably represents approximately the actual number of cases of hypertrophy of the heart which are really secondary to chronic renal disease. When these figures are added up, it is seen that no less than 295 (nearly 6%) of persons in these 5,000 had hypertrophied hearts, due apparently to hyperpiesis, and responsible for their death on account of heart failure, or of cardiac infarction, or of cerebral hæmorrhage. This condition seems to be increasing. If we take the fourth thousand, we find that 7·9% had hypertrophied hearts from this cause, and in the last thousand 9·4% were so affected. Of 500 insane patients examined *post mortem* over this same period of time, only 14 (2·8%) came in this category.

Congenital defects of the heart amount to about five or six per thousand.

Cardiac infarction was responsible for 82 cases (1·6%). A further 70 patients had coronary disease causing fibrosis in the heart muscle sufficiently extensive to be recorded, and at the last 2,000 post-mortem examinations we had a special category of "coronary atheroma of cardiac importance", under which 63 cases were placed.

Ante-mortem thrombi have been met with in the heart in 123 cases.

There were 78 aneurysms of great vessels, to which must be added 13 more specifically described as atheromatous aneurysms—that is, not associated with syphilitic disease.

There were 18 dissecting aneurysms—a condition which seems to be increasing. Syphilitic aortitis alone was met with 56 times; in 33 further cases it extended to the valve. Most of the 78 aneurysms were due to this cause. There were thus about 167 cases of syphilitic disease of the aorta altogether, or 3·3%.

Massive pulmonary embolism was present in 70 cases (1·4%). Infarcts were present in various organs, other than the heart, in 381 cases (7·6%).

Diseases of the Respiratory System.

In the respiratory system pneumonia is of course the important killing disease. Its contribution to death must be considered under five aspects. Lobar pneumonia is a distinct clinical and pathological entity, and in the cases recorded may be taken, almost without exception, as having been responsible for deaths in successive thousands as follows: 52, 34, 28, 26, 37 (177, or 3·5%). With modern drug treatment the number of these deaths will be much diminished in the future. The figures just given do not show any decline in the mortality rate (and this may be taken as parallel with the mortality rate as it was before treatment with sulphonamides came in) during the latter thousands.

Pneumonia directly due to virus diseases, such as psittacosis, form a second category, and that occurring as a complication in pneumonic influenza, measles, whooping cough and so on, a third. We have had no case of pneumonia in psittacosis, and the number of cases classified as influenzal totals 16.

The fourth category is that of bronchopneumonia occurring often in the later years of life and attributable to a preceding bronchitis or infection of the bronchial tree. I have tried to distinguish these cases from those of hypostatic pneumonia occurring as a happy release in advanced malignant disease, among bedridden old folk, in cases of cerebral injury and so on. Bronchopneumonia is an active process and in some of the cases was the primary cause of death; hypostatic pneumonia, on the other hand, though in many of the cases in which it occurred it was responsible for death, can be looked on as merely incidental. In some cases bronchopneumonia would appear on the death certificates as the registrable statistical cause of death, but not so the hypostatic pneumonia. Individual pathologists doubtless will differ to some extent as to the classification of their cases, and one's own opinion may swing one way or the other over a period of years. The cases of bronchopneumonia in successive thousands were as follows: 59, 64, 50, 41, 82 (296, 5·9%), and the cases of hypostatic pneumonia 44, 14, 49, 48, 38 (193, 3·8%). With bronchopneumonia must be placed those pneumonias which I have called atypical or irregular; some of course may have been true virus pneumonias, but probably nearly all were just diffuse or atypical bacterial pneumonias. These were not separated from the other pneumonias in the first thousand; the figures therefore are 17, 16, 34, 29 (96, nearly 2%).

If we add together the cases of bronchopneumonia and the cases of atypical or irregular pneumonia, these conditions being (apart from lobar pneumonia) important factors in the illnesses of the deceased persons and in many instances the cause of their deaths, we get the following figures: 59, 81, 66, 75, 111 (392, 7·8%). If we add the cases of lobar pneumonia, the figures are 111, 115, 94, 101, 148 (559, 11·2%). If we add still further the cases of hypostatic pneumonia, the figures are 155, 129, 143, 149, 186 (752, 15%). Thus 15% at least of the 5,000 subjects had pneumonia in one form or another at death.

The figures for septic and pyæmic pneumonia have remained low: 11, 5, 8, 6, 9 (39, nearly 0·8%).

Abscesses in the lungs are also few, but seem to have increased in number lately. The figures are 15, 13, 17, 12, 36 (93, 1·8%).

Silicosis occurs amongst old gold miners, in men who have worked at Broken Hill and occasionally in quarry workers near Adelaide. The term here includes miliary silicotic tubercles (often subpleural) as well as a number of cases in which there were large, stony-hard, dark grey masses in the upper lobes of both lungs, usually just above the centre, and smaller masses near the apices of the lower lobes. Some would call this tuberculo-silicosis; but in several instances in which material was ground up and injected into guinea-pigs no tuberculous lesions developed. In some cases there was, of course, unmistakable evidence of the presence of tuberculosis as well as silicosis; but in many of our cases the condition seems to be pure silicosis. If my interpretation is correct, the similarity of location to the lesions in adult pulmonary tuberculosis (though the lesions in the upper lobes are nearer to the centre of the lobes than in tuberculosis) would indicate that physical factors are responsible for the location of both the tubercle bacilli and the silica particles. Cases of silicosis have slightly increased in number recently; the figures are 12, 18, 12, 15, 25 (82, 1·6%).

Organizing or organized pneumonic exudates, giving rise to beefy or firm patches of lung tissue and sometimes converting a whole lobe into what looks almost like a beef steak, are commoner than is clinically suspected. The high figures for the first two thousands (if these are not due to errors in interpretation) are probably accounted for if viewed as aftermaths of the notorious pneumonic influenza epidemic of 1919. Twelve to eighteen per thousand probably represents accurately the present incidence of this disability in adults. The figures are 26 (possibly more), 40, 13, 12, 18 (109, 2·2%).

Fibrosis of the lungs, apart from silicosis and organized exudates, has remained low and constant in incidence. The figures are 6, 10, 7, 6, 6 (35, 0·7%).

As so many of our patients are elderly, it is not surprising that emphysema is common. In the first thousand, insufficient records of this lesion were tabulated, but thereafter the frequency with which it was found necessary to notice it in the summary was high. The figures are 18, 93, 140, 85, 98 (434, 8·7%). Probably 10% represents the real incidence of appreciable emphysema.

Diseases of the Alimentary System.

With regard to diseases of the alimentary system, gastric ulcers predominate in each thousand over duodenal ulcers (a few scars of old ulcers are included) and seem to be slightly on the increase. The figures for gastric ulcer in the successive thousands are 19, 23, 26, 25, 39 (132, 2·6%), and for duodenal ulcer, 12, 15, 19, 20, 27 (93, 1·85%).

Meckel's diverticulum is met with in from 1·7% to 2·5% in various thousands, and diverticulosis of the colon, sufficiently severe to appear in the summary, in about 3%.

Appendicitis was the cause of death in 12 and 15 cases respectively in the last two thousands of post-mortem examinations, paralytic ileus in 12 and 10 cases, and intestinal obstruction (other than that associated with malignant disease) in 16 and 34 cases respectively (this was due to various forms of hernia in 6 and 12 cases, and to bands or adhesions in 8 and 9 cases respectively).

Dysentery, ulcers (not malignant) in the colon and ulcerative colitis are relatively infrequent and seem to have decreased in incidence, the figures for all together being 16, 27, 23, 7, 3 (75, 1·5%).

Cases of carcinoma of the œsophagus numbered 9, 2, 11, 14, 7 (43, 0·86%); of carcinoma of the stomach, 37, 23, 37, 25, 28, 22 (172, 3·4%); of carcinoma of the colon, 10, 12, 16, 16, 17 (71, 1·4%); and of carcinoma of the rectum, 2, 9, 3, 10, 6 (30, 0·6%).

During the latter part of the period under review most persons who died from malignant disease were looked after elsewhere than at the Royal Adelaide Hospital. Those who died there thus mostly represent acute terminal emergencies. Consequently no inference can be drawn from the above figures as to whether malignant disease in these situations is or is not more prevalent in the later years.

Diseases of the Nervous System.

In the central nervous system the incidence of pyogenic leptomeningitis, in the absence of any outbreak of meningococcal meningitis, is a fairly uniform rate per thousand, namely 18, 29, 21, 15, 27 (110, 2·2%). The incidence of tuberculous meningitis is less than half of this, namely, 7, 13, 13, 6, 7 (46, 0·9%).

The incidence of berry aneurysms of the circle of Willis and pia-arachnoid hæmorrhage, including aneurysms which had not ruptured and hæmorrhages in which no aneurysms could be demonstrated, was as follows: 7, 8, 10, 10, 10 (45, 0·9%). This lesion in most cases was the cause of death.

Gliomata accounted for the following deaths: 11, 12, 19, 24, 9 (75 deaths, 1·5%). Secondary deposits in the brain or its membranes accounted for the following deaths: 5, 4, 6, 4, 6 (25, a proportion of 3 to 1).

Ordinary cerebral and cerebellar hæmorrhages unaccountably increased in the fourth thousand, but had fallen somewhat in the fifth. The figures are 41, 38, 36, 65, 52 (232, 4·6%).

The cases of cerebral softening are about the same in number, though it should be noted that whilst the cerebral hæmorrhage patients nearly always died from this condition, cerebral softenting was sometimes present in patients who died from quite unconnected causes. The numbers are 26, 42, 51, 57, 46 (222, 4·4%).

The number of deaths due to fractures of the skull and cerebral injuries depends on the decision of the coroner. Hence the variations. The figures are 67, 74, 26, 20, 42 (229, 4·5%).

Diseases of the Renal and Genito-Urinary System.

Congenital Anomalies of the Kidneys.

There were five examples of horseshoe kidney, four of them occurring in a single thousand post-mortem examinations. Another kidney was a compound kidney, and yet another was found in the sacral position.

Congenital Polycystic Kidneys.

The figures for congenital polycystic kidneys are 5, 5, 1, 5, 9 (25). They were not always associated with cysts in the liver, and when they were, only small cysts were present in that organ.

Large Simple Cysts of the Kidneys.

Large simple cysts are quite common, as these figures show: 6, 7, 18, 33, 17 (81). The figures comprise only cysts one inch in diameter or more. One would expect that from time to time a blow in the loin might rupture such a cyst, but if this does ever occur I have not heard of any clinical symptoms that might be attributable to it.

Uræmia.

Uræmia does not seem a common condition in South Australia, our figures being 7, 11, 20, 11, 11 (60).

Acute or Subacute Nephritis or Nephrosis.

The figures for acute or subacute nephritis or nephrosis are 13, 12, 5, 7, 18 (35, 1·1%). This heading, however, probably includes a number of conditions which were not true nephritis at all.

Chronic Nephritis.

My opinion of chronic nephritis has altered considerably over the period of twenty-five years. In the early series the part that essential hypertension plays in causing hypertrophy and failure of the heart was not fully recognized, and I felt compelled to read into the kidneys, as a cause of the hypertrophied heart, degrees of fibrosis which were either secondary to the generalized condition of arteriosclerosis or were relatively trivial. For instance, the figures for chronic nephritis, really meaning fibrosis of the kidneys, due to various causes, are 89, 58, 54, 35, 41, of which 63(?), 14, 27, 19, 18 might be described as considerable, the last three sets of figures representing probably the real situation. The figures for chronic nephritis and hypertrophied heart were 79, 68, 39, 29 and 20; of these cases, in 25, 21, 8, 12 and 8 the hearts were dilated and had failed. Here again the last-mentioned three figures probably represent the correct incidence of this condition. Figures for renal fibrosis and cerebral hæmorrhage, the total number for cerebral hæmorrhage being given in parentheses, are: 8 (41), 7 (39), 4 (36), 9 (65), 4 (52).

In "The Statistical Report of the State of South Australia for the Year 1942-1943", Part II, at page 6, return number 8 shows "The Number of Deaths and Rates per 10,000 of Mean Population from Certain Causes, 1915-1942". Nephritis was responsible for 267 deaths out of 6,712 from all causes in the year 1942, the rate per 10,000 being 4·39. The rate for all causes of death has risen slightly, from 96·8 in the five-year period 1920-1924 to 104·7 in 1941 and 110·3 in 1942. The rate for nephritis has varied from 3·91 in 1940 to 4·62 in the period 1930-1934. Over this period the rate per 10,000 for all deaths is somewhere about 100, and of those attributed to nephritis a little over four—that is, a little more than 4% of the deaths from all causes were attributed to nephritis on the death certificate. For statistical purposes deaths attributed to "nephritis" come under items 130, 131 and 132 of the "International List of Causes of Death—1938 Revision". Item 130 is "acute nephritis", item 131 is "chronic nephritis", item 132 is "nephritis not stated to be acute or chronic". Printed instructions are followed by the Registrar-General's Department in allocating to these three items various other terms that the medical man may have used in the certificate. Thus, under one or other of the above three headings would be placed "cardio-vascular renal disease", "exudative nephritis" and "uræmia", as well as a number of terms in which the word "nephritis" appears. In the case of "joint causes of death" certain rules have to be followed, as one or other of the joint causes finally has to be selected. The instructions are that "practically all heart diseases except acute and unspecified myocarditis" and "cerebral hæmorrhage, embolism and thrombosis, etc." are preferred to attributing death to items 130, 131 or 132 (nephritis). Death from nephritis, then, for these statistical purposes should consist almost entirely of death from uræmia. If the person had had primary chronic nephritis, which had caused hypertrophy of the heart, and had then died from failure of this hypertrophied heart, the death would be attributed to disease of the heart. Similarly, if death had been due to cerebral hæmorrhage following from the high blood pressure resulting from the renal disease, death would be attributed to cerebral hæmorrhage. My figures quoted above show that only about 1·1% of deaths of adults in the Royal Adelaide Hospital are due to uræmia. In return number 8 of the statistical report previously quoted, deaths from all causes will, of course, include also deaths of children, and children are almost excluded from my figures. If the deaths at the Royal Adelaide Hospital are, therefore, to be compared with deaths in the community at large, the ratio of about 1·1% of deaths from uræmia would, if all deaths were included (those of children as well as those of adults), be decidedly less. It therefore seems probable that the deaths in our statistical returns for "nephritis" are more than four times too numerous.

Infective Nephritis, Pyelitis and Abscesses in the Kidneys.

Infective nephritis, pyelitis and renal abscess appeared in 24, 38, 43, 52 and 60 cases (217). Renal or vesical

calculi were noted in 25, 17, 26, 21, 21 cases (110). The prostate was noted as enlarged in 20, 31, 45, 54, 64 (214) subjects.

Puerperal Conditions.

The figures for puerperal infections were 11, 22, 24, 16 and 11 (84). Of these, those due to *Clostridium welchii* or its allies were 6, 10, 14, 9, 5 (44). Only about seven infections were recognized as streptococcal and six were due to *Staphylococcus aureus*. Four deaths were due to tetanus. There were very few examples of toxæmia of pregnancy—5, 5, 4, 3, 1 (18); one of these was due to puerperal eclampsia.

Diseases of the Ductless Glands et Cetera.

The Suprarenals.

There were seven cases of Addison's disease; five of the subjects were tuberculous and two were not. In addition, there were two further cases of bilateral caseous tuberculosis and three cases of unilateral tuberculosis. There were three examples of massive bilateral hæmorrhages into the suprarenal glands; unilateral hæmorrhage occurred in one case, bilateral hæmorrhagic infarction in both suprarenals in two cases, and unilateral hæmorrhagic infarction in three. The figures for small cortical adenomata are 4, —, 7, 5, 7 (23).

Suprarenal glands are occasionally found plastered on the kidney in such a way that when the capsule is stripped off the kidney the cortex of the suprarenal is split in two, one half coming away with the capsule, the other remaining on the kidney. On microscopic examination it is seen that the suprarenal cells come into juxtaposition with the renal tubules without any connective tissue barrier between them. The number of instances in which this condition was noted is as follows: 1, 2, 1, 3, 2 (9).

Thyreoid Gland.

There were seven examples in which, in the course of a more or less generalized pyogenic infection, purulent infiltration or miliary abscesses were present in the thyreoid gland. There was actual myxœdema, or the thyreoid was fibrotic or atrophic, in 21 cases. Nodules or cysts were present in the thyreoid gland in between 1% and 2% of cases. There were only ten subjects who had thyreotoxicosis, including exophthalmic goitre.

Parathyreoid Gland.

There were only three examples in which the parathyreoid gland was enlarged.

Pituitary Gland.

There were three cases of acromegaly and one of Fröhlich's syndrome with a small, soft epitheliomatous growth in the *sella turcica*.

Thymus Gland.

The figures in the last 4,000 autopsies for persistence of the thymus gland were as follows: 7, 10, 4, 5 (26). One of the subjects was a woman, aged thirty-seven years, and another was a man, aged twenty-six years.

Pineal Gland.

The pineal gland was noted as calcified in one case and cystic in another.

The Spleen.

An enlarged spleen due to relapsing fever was found in a Lascar admitted to hospital from on board ship. There were two examples of ruptured spleen and one of an abscess in the spleen.

Blood Diseases.

Pernicious anæmia has decreased, but cases are still occurring in which the condition is resistant to treatment or the patient comes too late. The figures are 10, 7, 5, 4, 6 (32). The figures for leuchæmia are interesting, showing apparently an increase with a present incidence of about 2%. The figures are 7, 5, 21, 16, 19 (68). The figures for Hodgkin's disease or allied conditions were 3, 1, 7, 6, 3 (20). About one or two cases per 1,000 of purpura or purpuric conditions are met with. Two men died from acute hæmolysis of unknown origin, which developed while they were on an oil-tanker, just after leaving port. The figures for hæmochromatosis are —, ?2, 5, 3, 1 (11).

Malignant Disease.

The number of deaths from cancer at the Royal Adelaide Hospital can no longer be taken as a guide to the incidence of the various forms of cancer in the community at large. During the course of the first 2,000 post-mortem examinations a cancer block was still attached to the Adelaide Hospital. Later these "hopeless cases" were removed elsewhere. The patients now treated at the Royal Adelaide Hospital are those in the early stages, when treatment may be of some avail, or those who suffer from serious incidents, such as intestinal obstruction, during the progress of the disease. The total number of cases of carcinoma in the 5,000 autopsies was 700, of gliomata 75, of sarcomata 32, and of malignant melanomata 10. The following figures show the incidence of carcinoma in various sites: œsophagus, 9, 2, 11, 14, 7 (43); stomach, 37, 23, 37, 28, 22 (147); colon and rectum, 12, 21, 19, 26, 23 (101); pancreas, 5, 7, 9, 5, 4 (30); lung, 2, 3, 10, 18, 22 (55); prostate, 6, 9, 9, 7, 15 (46); malignant melanomata, 2, 0, 5, 9, 1, 2 (19).

From these figures it will be noted that there seems to be little change in the incidence of carcinoma of the œsophagus, that of carcinoma of the stomach seems to be decreasing, whilst that of carcinoma of the large intestine seems to have remained more or less stationary. A definite increase in the incidence of carcinoma of the prostate seems to have been observed during the last 1,000 post-mortem examinations, perhaps to be attributed to the increasing age of the population. There is an unquestioned increase in carcinoma of the lung.

Infectious Diseases.

Pulmonary and Other Forms of Tuberculosis.

During the first 2,000 post-mortem examinations the Consumptive Home was attached to the Adelaide Hospital and the deaths from pulmonary tuberculosis probably show a ratio equivalent to that in the hospital population at large. Since then a special hospital deals with most of the cases of pulmonary tuberculosis, and the deaths from this disease at the Royal Adelaide Hospital are more or less those of "emergency" patients or of patients whose condition has not been recognized clinically. The figures for pulmonary tuberculosis, as the cause of death or of considerable degree, are 73, 92, 53, 21, 29 (268, 5·3%). In addition the following figures represent slight degrees of pulmonary tuberculosis, but do not include healed lesions: 29, 23, 17, 14, 20 (103, 2%). Patients suffering from tuberculosis, other than pulmonary tuberculosis, still come to the Royal Adelaide Hospital, and the following figures show that its incidence has been more or less uniform throughout the period of twenty-three years: 23, 32, 26, 19, 21 (121, 2·4%). The figures for caseous or calcified mesenteric glands also remained fairly uniform, thus: 10, 11, 9, 20, 14 (64, 1·3%).

Other Infectious Diseases.

Only three cases of actinomycosis have been recorded. Very few deaths now occur from typhoid fever; only one post-mortem examination was performed on such a subject in the last 2,000 autopsies. The figures for the series are 10, 6, 4, —, 1 (21).

Three fatal Salmonella infections occurred. There were eleven fatal cases of diphtheria, four of measles, and three of whooping cough, and one child died after chicken-pox. There was one case of anthrax (not diagnosed clinically), one case in which a Brucella infection had occurred, and five patients who had had endemic typhus. The figures for tetanus are small, namely, 2, 5, 11, 5, 1 (24); these show a slight increase followed by a decrease. There was a case of *periarteritis nodosa* and one case of chorea.

Generalized Pyogenic Infections.

The figures for pyæmia, cellulitis *et cetera* were 20, 37, 28, 33, 32 (150). During the last 3,000 *post mortem* examinations *Staphylococcus aureus* infections, comprised in

the above figures, constituted 14, 17 and 18—that is, between 1% and 2% of all the patients have died from *Staphylococcus aureus* infections.

Helminthic and Protozoal Infestations.

Hydatid Cysts.

Hydatid cysts, mostly degenerated, are still met with at more than 1% of post-mortem examinations. During the first 3,000 post-mortem examinations they were found in 2% or over, but the figures have dropped recently. The numbers are 23, 20, 26, 13, 14 (96, nearly 2%). Of these the hydatid cysts were responsible for death in 5, 6, 6, 0, 0 (17 cases).

Other Helminth Infestations.

South Australia is probably an area in the world where human beings are least afflicted with helminth infestations, if hydatid cysts are excluded. The practical absence of an indigenous native population, a hot, dry climate with its long summer, and the high standard of living are all contributory factors. Hookworms were found in only one subject, a coloured person from overseas; *Oxyuris* was found in one, though probably often overlooked in others; *Ascaris* was found in two and *Tæniæ saginata* in one.

Protozoal Infestations.

Malaria does not occur naturally in South Australia; the only case was one of introduced malaria in a general paralytic. Amœbic dysentery occurs occasionally in South Australia, but only three examples were met with at the Royal Adelaide Hospital, compared with 18 found at 600 post-mortem examinations on patients at the mental hospitals.

Nutritional and Various Conditions.

From one to six cases of gout are met with in each 1,000 post-mortem examinations. Amyloid disease is uncommon now, the figures being 2, 6, 7, 2, 1 (18). There were three examples of heat stroke or heat exhaustion. The figures for extensive bed-sores during the last 4,000 post-mortem examinations were —, 26, 24, 7, 16 (73). Transposition of the viscera occurred twice.

The figures for death associated with anæsthesia, but not always due to it, were 7, 3, 12, 3, 9 (34); one of these deaths was due to air embolism. The figures for deaths from poisoning were 7, 9, 8, 8, 9 (41).

Accidents.

The number of post-mortem examinations performed on accident victims has decreased, obviously not because accidents are fewer, but because the coroner has not required so many to be made. The figures are 121, 113, 56, 50, 68 (408).

No Lesions of Importance or Causes of Death Obscure.

I find myself not infrequently, in spite of the utmost care, unable to find a reasonable cause for the patient's death, or any lesions of importance. It will be seen from the following figures—4, 6, 14, 14, 25 (63)—how difficult it has been found in some cases to account for the patient's death. It almost seems as if with increased experience one finds it more difficult to determine the nature of the condition. It will be seen that from 1·4% to 2·5% of our post-mortem findings come in this category. These cases would probably well repay a closer analysis. Various nutritional conditions, unaccompanied by a reasonable history, might readily be overlooked at the post-mortem examination. Cases of poisoning in various ways also might not be recognized unless there was some clue to their nature.

Wassermann Reactions.

A record has been kept of the Wassermann reaction when this test has been performed. These cases have been tabulated separately, so that one can see how many subjects reacting to the Wassermann test were found on post-mortem examination to have lesions that could be referred to syphilis, as well as of the lesions that were present in the persons who failed to react to the test. This record was started only in the middle of the second thousand post-mortem examinations. The figures for reactions to the Wassermann test are 19, 30, 27, 23 (99); the figures for failure to react to the Wassermann test were 51, 142, 130, 61 (384). The number of Wassermann tests carried out was greatly reduced during the last 1,000 post-mortem examinations on account of the war. It should be remembered that the Wassermann test is not performed as a routine measure on every patient who comes into the hospital, but only in those cases in which there is perhaps a possibility that syphilis has been contracted. The figures, as far as they go, seem to indicate that syphilis is not a prevalent disease in South Australia.

The Killing Diseases.

The attached table (Table II) shows the conditions mostly responsible for death, first of all arranged according to systems and then in more detail. Malignant growths (including gliomata) head the list; but their incidence is perhaps exaggerated under hospital conditions. They total 856 (17·1%). Next come accidents (not all fatal) with 408 (8·2%). Cerebral and cerebellar hæmorrhages contribute 232 (4·6%). Pulmonary tuberculosis, mostly fatal, provides 268 (5·3%). Hypertrophy and dilatation of the heart, apparently attributable to hyperpiesis, and ending in heart failure are responsible for 196 (4%). Rheumatic valvular disease contributes 149 (3%). Bacterial endocarditis yields 112 (2·2%). Cardiac infarction produces 82 (1·6%), to which should probably be added the 70 cases of fibrosis of the cardiac muscle and other deaths from coronoray atheroma without these sequelæ; the deaths from coronary atheroma and its consequences in this series probably exceed 4% and appear to be increasing in frequency. Lobar pneumonia contributes 177 cases (3·5%). In a considerable proportion of the 382 cases (7·6%) of bronchopneumonia and atypical pneumonia these conditions were probably not statistically the cause of death but secondary to other conditions. It will be seen that uræmia taken as an index of Bright's disease occupies a very low place in the killing diseases, there being only 60 cases (1·2%); in addition to renal disease causing uræmia, in a small number of cases in our series renal disease was directly responsible for cardiac hypertrophy and eventual failure or for high blood pressure leading to cerebral hæmorrhage.

In "The Statistical Register of the State of South Australia for the Year 1942-1943", Part II, "Population and Vital", return number 8 gives the deaths from certain causes and the rates per 10,000 of mean population. If for

TABLE II.

The Killing Diseases.

Condition.	Number.	Percentage.
Malignant growths (nearly all fatal)	856	17·1
Major cardio-vascular diseases (mostly fatal)	764	15·3
Pneumonia (not all fatal)	559	11·2
Cerebral vascular lesions (not all fatal)	499	10·0
Accidents (some not fatal)	408	8·2
Pulmonary tuberculosis (mostly fatal)	268	5·3
Uræmia (as an index of primary renal disease)	60	1·2
Major cardio-vascular diseases:		
Hypertrophied and dilated hearts, apparently attributable to hyperpiesis, heart failure	196	4·0
Old rheumatic valvular defects (many fatal)	149	3·0
Bacterial endocarditis (all fatal)	112	2·2
Cardiac infarction (all fatal)	82	1·6
Fibrosis of cardiac muscle (many fatal)	70	1·4
Syphilitic aneurysms and aortitis extending to the valve	111	2·2
Total	764	15·3
Cerebral vascular lesions:		
Cerebral and cerebellar hæmorrhages (all fatal)	232	4·6
Cerebral softening (many not actual cause of death)	222	4·4
Pia-arachnoid hæmorrhages	45	0·9
Total	499	10·0
Pneumonia:		
Lobar pneumonia (all fatal)	177	3·5
Bronchopneumonia and atypical pneumonia (some fatal, others secondary)	382	7·6
Total	559	

convenience the year 1941 is taken, in which the rate for all causes was 104·7 (that is, approximately 100) we find that cerebral hæmorrhage, softening *et cetera* contribute (approximately) 9·84%; pneumonia (all forms), 6·85%; tuberculosis (all kinds), 3·7% (including respiratory, 3·41%); cancer, 12·64%; diseases of the heart, 26·32%; nephritis, 4·55%; and violence *et cetera*, 5·7%. A consideration of return number 12, in which diseases of the circulatory system are considered, makes it hard to see under what category the large number of deaths from failure of hypertrophied hearts, apparently due to hypertension and without adequate valvular or renal cause, are placed. Of the 1,665 cases of heart disease, 852 appear under "diseases of myocardium—other myocardial degeneration" and 368 under "diseases of coronary arteries", a total of 1,220 or three-quarters of the whole. Evidently many of these hypertrophied hearts from hypertension have been placed under "other myocardial degenerations", as the coronary artery cases seem well provided for. There appears an insufficient number of deaths from valvular disease (157) and bacterial endocarditis (7). The number of 244 deaths from chronic nephritis seems much too high.

Summary.

1. The pathological lesions found at 5,000 Australian autopsies carried out between 1920 and 1943 have been tabulated. The method of tabulation is described. This method shows in each case under its appropriate heading the lesion itself, the age and sex of the patient, and all other pathological lesions of significance.

2. The results are published at the conclusion of each thousand in *The Medical and Scientific Archives of the Royal Adelaide Hospital.* The information is thus available to the medical profession. By tabulation in successive thousands changes in the incidence of disease can be shown.

3. The most important causes of death apart from malignant growths, pulmonary tuberculosis and accidents are: failure of hypertrophied and dilated hearts attributable to hyperpiesis (4%), coronary disease and its consequences (about 4%), cerebral and cerebellar hæmorrhage (about 4·6%), and lobar pneumonia (3·5%).

4. It would appear that in the official statistics for deaths, the heading which should include hypertrophied and dilated hearts from essential hypertension is not provided for, and that the number of deaths from nephritis is excessive.

A SURVEY OF SCRUB TYPHUS (K TYPHUS) IN NEW GUINEA.

By J. DE VIDAS,
Adelaide.

SCRUB TYPHUS in the New Guinea area falls into the same disease group as tsutsugamushi fever in Japan, Formosa and Korea, rural typhus in Malaya, and Sumatran mite fever. Lewthwaite and Savoor[1] have pointed out the common relationship of these diseases, in which the causal organism is *Rickettsia orientalis* or one of its strains, the animal reservoir a field rat, and the arthropod vector a larval mite. Their clinico-pathological picture conforms more or less to the same pattern, and the ecological and epidemiological background of disease is fairly constant.

Definition.

Scrub (mite-borne or K) typhus is an acute febrile disease, which is, as a rule, severe and sometimes fatal. It is characterized by (i) a high remittent fever with the symptoms and signs of an acute infective process (headache, malaise, general aches and pains, gastro-intestinal disturbances and prostration), (ii) an eschar at the site of the arthropod's bite, (iii) a maculopapular rash appearing at the end of the first week of the illness, and (iv) generalized lymphadenopathy. The course of the illness varies, depending on the general resistance of the patient and the locality of infection; it may end in death at the end of the second week in a fulminating infection, in partial recovery at the end of the third week followed later by sudden death from cardiac failure, or in complete recovery after a prolonged convalescence.

Ecology and Epidemiology.

A locality of infection presumably depends on the existence of the rat-mite-rat cycle. This would be maintained by local ecological factors conducive to the developmental demands of the arthropods and animal reservoirs that harbour the rickettsial organism. Thus areas of scrub typhus infection are found in (i) the open prairies of kunai grass and rain-forest areas of the d'Entrecasteaux Group, (ii) the untended plantations and jungle growth of the Schouten and Purdy Groups. In these areas, wet humus for the continued existence of larval mites is present, together with food and shelter for the rats. (Larval mites may be regarded as minute spheres in which, as the radius tends towards zero, the surface area diminishes less rapidly than the volume; consequently the larval mite requires a wet environment to replace its fluid loss in order to maintain life.)

The distribution of these areas of infection is characteristically patchy. Furthermore, the virulence of infection as indicated by the case mortality varies with different localities. This focal distribution of disease and virulence may be related to the specificity of the larval mite and the organismal strain. For example, the species of larval mites found in the scrub typhus areas of Goodenough Island differ from those occurring in nearby "scrub-itch" areas.[2] That only certain species of larval mites carry infection is still *sub judice.* But the general consensus of opinion is that *Trombicula fletcheri* and *Trombicula walchi* transmit scrub typhus, while *Trombicula mediocris, Schöngastia pusilla* and *Schöngastia blestowei* produce "scrub-itch". However, more experimental work needs to be done in confirming the recovery of rickettsial strains in larval mites from boot collections and rats and in animal reservoirs.

Clinico-Pathological Picture.

The disease has a fairly constant clinical and pathological pattern. Isolated cases of infection during a campaign are not common, so that a group-pattern of disease usually emerges in a particular area. Diagnosis is therefore relatively easy; if there is any doubt, then the Weil-Felix test will later give decisive evidence. The slight variations among cases, as will be seen later, are due to differences in severity and duration of the illness. The following examples will illustrate the usual clinico-pathological picture.

CASE I.—This patient was admitted to hospital with an acute febrile illness of four days' duration. He was suffering from headache, generalized aches and pains, malaise, and gastro-intestinal disturbances. On examination, he was found to have a temperature of 103·4° F. An eschar was present on the back of the right knee, together with a maculopapular rash over the arms, shoulders and trunk, and a generalized enlargement and tenderness of the lymph glands. The symptoms became worse and the patient was prostrated. Two days later a productive cough developed, the respiration and pulse rates were increased, and adventitious sounds were heard in the lungs. The spleen was palpable and tender. The patient's condition rapidly deteriorated, and he became stuporose and delirious. Twelve days after the onset of the illness, death occurred with hyperpyrexia, a greatly increased respiration rate and a fast, thready pulse.

The laboratory findings were as follows. The hæmoglobin level and red blood cell count were normal, and the white blood cells numbered 10,700 per cubic millimetre, a "shift to the left" being present. Albumin was found in the urine. Examination of blood films for malaria parasites gave negative results.

Treatment was symptomatic and supportive; it consisted in the intravenous administration of plasma and glucose and saline solution, the exhibition of morphine, and the giving of "Prostigmin" to relieve abdominal distension. Sulphadiazine, and thirty cubic centimetres of blood from a person who had recovered from scrub typhus six months before, were administered without effect by the oral and intramuscular routes.

A post-mortem examination was carried out. The lungs were affected by advanced passive congestion with dark-red firmer areas. The blood vessels lining the alveolar walls were dilated, and red cells filled the alveolar spaces. Small collections of lymphocytes and histiocytes were seen round the vessel walls and inside the alveoli.

FIGURE I.

Perivascular collection of inflammatory round cells in scrub typhus. The arteriolar wall is still intact. (High power of the microscope; hæmatoxylin and eosin stain.)

In the heart a few subpericardial hæmorrhages and dilatation of small blood vessels were found. The heart was enlarged and flabby, the myocardium being grey-red and softer than normal. The coronary arteries, valves and endocardium were all normal. Microscopic examination showed the vessels in the subpericardial fat and myocardium

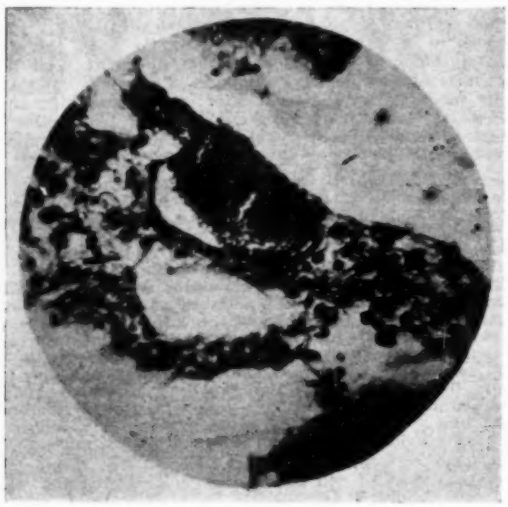

FIGURE II.

Infiltration of the meninges by histiocytes and lymphocytes, and perivasculitis. (High power of the microscope; hæmatoxylin and eosin stain.)

to be engorged and surrounded by focal collections of lymphocytes and histiocytes and occasional plasma cells. These inflammatory cells became scarcer and more diffused in the intramuscular spaces. In the myocardium itself no appreciable damage was observed.

The spleen was enlarged to three times its normal size. It was purplish-red and mottled in appearance. The pulp was dark red and diffluent, and the normal markings were indistinct and the Malpighian corpuscles small. On microscopic examination, only a few normal trabeculæ were seen; only the outlines of Malpighian corpuscles were defined, although no change was detected in the central arteries. Increased cellularity, especially in the cells of the monocytic series, and erythrophagia were present.

The liver was enlarged and congested, and had an early "nutmeg" appearance. Examination of sections revealed a few inflammatory round cells beneath the capsule. A few small foci of lymphocytes and histiocytes were seen about the portal vessels.

The lymph glands were enlarged and soft, and had a pale medulla and a yellow cortex. All the lymph follicles were indistinct, and the lymph channels were stuffed with mononuclear cells, which constituted to a large degree the increased cellularity of the organ.

The kidneys were congested, and small petechiæ were present in the mucosa of the pelvis. Congested glomeruli and engorged pyramidal vessels were seen, and the smaller vessels were surrounded by inflammatory mononuclear cells.

FIGURE III.

Brain-stem lesion in scrub typhus, showing scanty collections of inflammatory round cells at junction of vessel wall and Virchow-Robin space. (High power of the microscope; hæmatoxylin and eosin stain.)

The brain was congested. There were a few areas of round-cell infiltration about the meningeal vessels; very little change was present in the brain substance and, when seen, focal and perivascular round-cell inflammation was scanty.

The testes were soft; increased numbers of round cells surrounded the seminiferous tubules and were present in the interstitial tissue about the vessels.

The suprarenals were congested.

Coagulation necrosis of the base of the eschar was present, together with many lymphocytes and histiocytes. Similar collections were present round the sweat glands and hair follicles.

CASE II.—The patient was admitted to hospital with a history of sudden onset of chills, malaise, gastro-intestinal disturbances and feverishness. On examination of the patient, his temperature was 102° F. and his pulse rate 80 per minute; the systolic blood pressure was 180 millimetres of mercury and the diastolic pressure 60. There was a maculo-papular rash on the body, and the superficial lymph glands were enlarged and tender. No eschar was seen. The patient's condition rapidly deteriorated, the temperature rising to 105° F., the pulse rate to 130 per minute and the number of respirations to 36 per minute. Signs of consolidation in the lungs appeared, and death occurred after an illness lasting thirteen days, the patient being in a state of semi-coma.

The laboratory findings were as follows. The hæmoglobin value was 85%; the red blood cells numbered 4,600,000 per cubic millimetre and the white blood cells 7,200 per cubic millimetre (stab cells 28%, polymorphonuclear cells 58%, lymphocytes 14%). Albumin was present in the urine.

The treatment was symptomatic and supportive.

A post-mortem examination was carried out. The lungs were dark red posteriorly, and the cut surface exuded a sanious fluid; in other places the lungs were pale grey and carnified. On microscopic examination the small and large vessels were congested. Inside the alveoli were hæmorrhages, œdema and eosinophilic material. Some of the cells lining the alveoli were swollen, while the alveolar walls contained many dilated blood vessels, histiocytes, lymphocytes and occasional polymorphonuclear cells. A small amount of fibrin was present in a few alveoli.

Examination of the heart revealed subpericardial hæmorrhages and the myocardium was pale and flabby. The coronary vessels, valves and endocardium were normal. In the subpericardial fat there was great congestion of the vessels, and about them and the mesothelial surface were collections of lymphocytes and histiocytes. Similar collections were present between the muscle cells in the left

FIGURE IV.

Portal infiltration of liver by inflammatory round cells in scrub typhus. (High power of the microscope; hæmatoxylin and eosin stain.)

auricle, the upper part of the left ventricle, and root of the mitral valve. Towards the apex they became scantier. Examination of most of the muscle fibres showed cross-striation, but the cytoplasm of some was homogeneous. Œdema of the intercellular spaces was greatest near the areas of cell infiltration.

The spleen was about three times larger than normal. Its cut surface was red purple and its architectural pattern indistinct. On microscopic examination the pulp was seen to be congested. Some erythrophagia and occasional polymorphonuclear cells were present, but the bulk of the cells belonged to the monocytic series. The trabeculæ were scarce, and the outlines of the Malpighian bodies were indistinct.

The liver was enlarged and presented an early "nutmeg" appearance. In some portal areas there was a slight increase of inflammatory round cells. Congestion and dilatation of the small vessels and sinusoids with small hæmorrhages into the tissues were present. The polygonal cells contained pink granules, but were otherwise normal.

Examination of the kidneys revealed congested glomeruli and vessels, and cloudy swelling of the tubule cells. Only small collections of round cells were seen.

Little change was found in the suprarenals, except for congestion of the vessels and a moderate number of mononuclear leucocytes, and infiltration of the periadrenal fat by lymphocytes and histiocytes.

Case Analysis.

In this series the mortality rate was 20 cases in 80 over the whole period. More deaths occurred in the first half of that period than in the second half, for the obvious

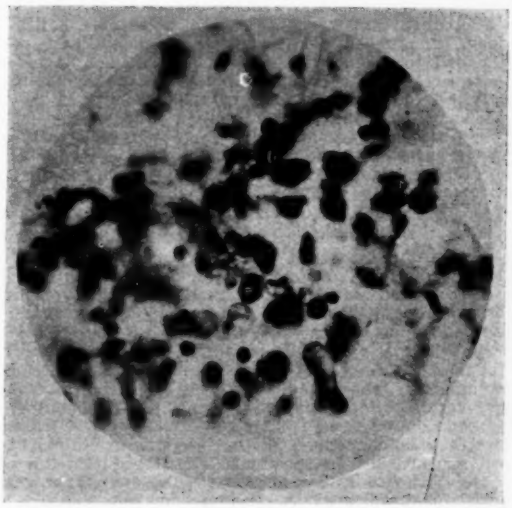

FIGURE V.

Detail of inflammatory cells (histiocytes and lymphocytes) illustrated in Figure IV. (Oil immersion lens; hæmatoxylin and eosin stain.)

reasons that localities of infection were cleared, personnel became "mite-conscious", and full recognition of the disease provided immediate admission to hospital and absolute rest for the patient.

The analysis in Table I is made from 15 post-mortem examinations. These figures further illustrate the constancy of the disease pattern.

Of 15 cases, an eschar occurred in 11, a rash in 11, and adenopathy and splenomegaly in all.

TABLE I.

Observation.	Mean Value.	Standard Deviation.
Age (years)	26	2·5
Duration of illness in days	15	3·0
Temperature on admission to hospital	102° F.	1·1° F.
Number of leucocytes per cubic millimetre of blood	10,300	3,900

Severe myocarditis was present in nine cases, and less severe myocarditis in six; included in these were three cases of hydropericardium. Bronchopneumonia occurred in seven cases, and severe congestion and œdema of the lungs in eight; included in these were seven cases of hydrothorax. In all 15 cases the kidneys were affected only by cloudy swelling and congestion, although in three cases in addition hæmorrhages were found in the pelvic mucosa. Similarly, in all cases congestion was present in the brain, and in three also petechial hæmorrhages in the meninges were observed. Cloudy swelling and congestion of the liver appeared in all cases, and fatty degeneration was present in three cases.

Histopathological Findings.

The histopathological lesions in these cases were fairly constant. There was predominantly a perivascular round-cell infiltration in the vital organs, especially the lungs, heart, liver, kidneys, spleen and lymph glands. The inflammatory round cells consisted of lymphocytes, histiocytes and an occasional plasma cell. Their greatest concentration

was round the small blood vessels, and they tended to thin out when traced outward into the tissue substance.

Swelling of the vascular endothelium with a tendency to thrombus formation, as described by Corbett,[3] was not the rule in this series. On the other hand, examination of tissue sections obtained elsewhere from two patients who died about six weeks after the onset of the illness, revealed pronounced perivasculitis, vasculitis and endothelial proliferation, with thrombosis, hæmorrhage and tissue necrosis. Such a difference is, in my opinion, directly related not so much to the severity as to the duration of the illness. In other words, the histopathological events march in time with the disease, becoming arrested if the patient dies early, or progressing as the illness smoulders. It is not yet known whether the lesions resolve in those patients who recover, or whether permanent tissue changes remain as a result of an irreversible process.

In a preliminary report from the Office of the United States of America Typhus Commission[4] it is stated that, although endovascular thrombosis and hæmorrhage occurred in scrub typhus, it was conspicuously less than in classical typhus. It appears that the essential difference between the histogenesis of classical typhus and that of scrub typhus lies in the fact that the inflammatory spread is centrifugal from the inside of the vessel in classical typhus, whereas in scrub typhus the spread is centripetal from the outside of the vessel. This centripetal spread in scrub typhus may be demonstrated in the different histopathological appearances described above in those patients who died after a short illness, and those who died after a long illness. In the first series the infective process was too overwhelming and rapid to allow time for the histopathological changes fully to develop. The inflammatory spread from the periphery to the centre of the vessel was cut short before its conclusion by the early death of the patient. In these cases the tissue changes seen microscopically did not seem sufficient to cause death. It must therefore be presumed that the rickettsial toxins had brought about cellular death before the grosser histopathological signs could become manifest. On the other hand, in the other group of cases, the panvasculitis, thrombosis, hæmorrhage and necrosis of tissues in the vital organs were sufficient to warrant the death of the patient.

Serological Findings.

In a series of 80 Weil-Felix tests, Proteus OX-K being used as an antigen, the composite graph formed by plotting the titres during the course of illness is shown in Figure VI. It will be seen that there was no significant titre (a titre of 1 in 160 or over was regarded as significant) in the first ten days. Then the titre rose till it reached its maximum at the end of the third week. It continued there on a high plateau for about a week and then dropped during convalescence, finally to disappear in two months.

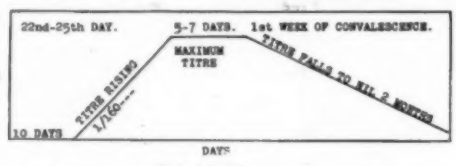

FIGURE VI.

Composite graph of results of eighty Weil-Felix tests.

The maximum titre bore no relation to the severity of the illness nor to its prognosis. Patients with a high titre died as readily as those with a low titre. The maximum titre appeared to vary with the individual. Léon and Apodaca[5] came to a similar conclusion from experimental work. There was no evidence that the antibody response as measured by the Weil-Felix test bore any obvious relation to immunity.

I have recently carried out a rapid slide agglutination test with whole dried patient's blood and concentrated Proteus OX-K antigen. The method was based on Bardhan, Tyagi and Boutros's[6] modification of Steuer's rapid slide test for classical typhus. A drop of patient's blood was allowed to dry on a glass slide and two drops of concentrated Proteus OX-K suspension were added to it. Another drop of suspension was placed elsewhere on the slide as a control. After having stood for one minute, the slide was gently rocked for five to ten minutes. A positive reaction was shown by the appearance of white granules. Each reaction was checked by a tube agglutination test. The results of 20 tests are shown in Table II.

TABLE II.

Weil-Felix Test Titre.	Result of Whole Blood Slide-Agglutination Test.
1 in 20	Negative.
1 in 1,280	Positive.
1 in 640	Positive.
1 in 160	Positive.
1 in 20	Negative.
1 in 80	Positive.
1 in 40	Negative.
1 in 640	Positive.
1 in 320	Positive.
1 in 640	Positive.
1 in 1,280	Positive.
1 in 640	Positive.
1 in 1,280	Positive.
1 in 1,280	Positive.
1 in 1,280	Positive.
1 in 1,280	Positive.
1 in 160	Positive.
1 in 1,280	Positive.
1 in 1,280	Positive.
1 in 320	Positive.

It will be seen that those specimens of blood with a significant titre all gave positive reactions in the slide test. As has been mentioned above, a titre of 1 in 160 was regarded as positive; a titre of 1 in 80 was considered doubtful.

The blood of five healthy persons was tested by the rapid slide method; all results were negative. Although it is obvious that all positive slide reactions must be checked by a tube agglutination test, this simple test may be of value in confirming the clinical diagnosis of an outbreak of scrub typhus in advanced field units.

Summary.

1. A brief survey of scrub typhus during the New Guinea campaign is given.

2. The various ecological factors and background of the disease are described.

3. The histogenesis of scrub typhus lesions is discussed.

4. The serology of scrub typhus and a new rapid slide agglutination test with whole dried blood and a concentrated Proteus OX-K antigen are described.

Acknowledgements.

My thanks are due to Lieutenant-Colonel Lippard, to Captain McEndie, to Lieutenant Montanaro and to Captain Rappaport for their help and information; and to Air Vice-Marshal T. E. V. Hurley for permission to publish this paper.

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THE PROPHYLACTIC USE OF SULPHANILAMIDE IN RHEUMATIC FEVER: PRELIMINARY REPORT.[1]

By S. G. ANDERSON, M.B., B.S.,
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COBURN[1] has remarked upon the parallel incidence of rheumatic fever and of hæmolytic streptococci in the throat. The possible ætiological relationship between these two phenomena has been considered and the suggestion made that the pathology of rheumatic fever depends on an allergic response to some product of the β-hæmolytic streptococcus group A, or of the body when affected by that streptococcus. In support of this theory it was noted that such streptococcal infections often preceded rheumatic episodes by periods varying from several days to several weeks.

It was argued, therefore, that if it was possible to prevent such streptococcal infections of patients with a rheumatic diathesis, rheumatic fever would not occur. Attempts have been made to reduce the incidence of group A streptococcal infections by maintaining in the blood a low but relatively constant level of sulphanilamide for long periods; it will be seen that, because of the usual latent period of one to three weeks between throat infections and rheumatic attacks, exhibition of sulphanilamide might not be expected to prevent rheumatic recurrences during the first three weeks of the treatment.

In the literature available at present, this type of prophylaxis has been reported only from America, and Dodge *et alii*[2] in a recent article reviewed the progress that has been made in this direction, and added cases of their own.

In August, 1944, with the cooperation of the honorary medical staff, we established at this hospital, as a branch of the rheumatic heart clinic, a section which has been concerned with the distribution of sulphanilamide and the supervision of those patients suffering from rheumatic fever who were taking the drug.

Selection of Patients.

The patients who were selected for prophylactic treatment with sulphanilamide had all suffered an attack of rheumatic fever in one of its forms, one or more times during the preceding five years, usually during the previous two years. Such patients varied in age from three to fourteen years. An attempt was made to exclude those few patients who would not have been cooperative, because of the distance of their homes from the city, because of mental deficiency of their parents, or for other reasons. The number so eliminated was small. With the exercise of no further choice than this, patients were accepted as they presented at the out-patient clinic or as they became sufficiently convalescent as in-patients.

Time of Commencing Therapy.

In the first few months after the institution of this treatment, some patients commenced treatment with sulphanilamide within five weeks of having been admitted to hospital with an acute attack of rheumatic fever. During these months it was found that in two cases, in each of which the initial attack had occurred less than three months previously, a recrudescence of the fever and rheumatic manifestations took place within ten days of the institution of sulphanilamide therapy. In view of the writings from overseas, it was thought that the sulphanilamide probably precipitated these early recurrences by having been given too early during convalescence. Therefore in the later months sulphanilamide prophylaxis was not inaugurated until the patients had attended the out-patient clinic for at least two months; this latter routine meant that the drug was not given to a patient within five months of the onset of his most recent acute attack of fever.

Dosage.

Children weighing under three stone were given 0·5 gramme per day in one dose; children weighing over three stone were given 0·5 gramme twice a day. For a few of the older and heavier children, 0·5 gramme was necessary three times a day.

Supervision.

It was considered advisable to examine these patients carefully and at frequent intervals, bearing in mind particularly two possible sequelæ of the administration of the drug—agranulocytosis and anæmia. The occurrence of one fatal case of the former disease persuaded Stowell and Button[4] in America to cease similar treatment in 32 cases. Therefore, at our clinic, every patient beginning to take sulphanilamide was examined once a week during the first, second, fifth and sixth weeks, and twice during both the third and the fourth weeks; thereafter the patient was examined once every month or six weeks. At each visit an estimation of the number of white blood cells per cubic millimetre of blood was made and recorded, as well as the hæmoglobin value of the blood. Then the patient was examined briefly by the same resident medical officer at each visit, and if necessary referred by him for investigation or for discussion and advice by other members of the clinic. In this way, it was found possible to form a comparative estimate of the patient's condition from week to week.

Number of Patients taking Sulphanilamide.

The administration of sulphanilamide was commenced early in August, 1944, eleven patients being given the drug initially. The following week, sixteen more children were included in the scheme, and the number was thereafter rapidly increased. February, 1945, saw 160 children taking the drug.

Of these 160, 123 children had suffered from rheumatic arthritis and carditis clinically demonstrated, but not from chorea; 12 had shown evidence of chorea in their past history, in addition to arthritis and carditis; and 21 had been diagnosed as suffering from chorea, but had never complained of swollen joints or muscle pains, though of this last-mentioned group 12 showed clinical evidence of carditis. The four remaining children had experienced attacks of rheumatic arthritis, but their records stated that at all times their hearts had been clinically normal.

Allowance being made for a latent period of three weeks at the beginning of treatment, during which time, it was believed, the drug might not prevent a recurrence of the fever, the number of children treated was estimated to be equivalent to 104 patients each of whom had been under treatment and observation for six months; the clinic is being continued at the time of writing.

Results.

Recurrences.

In the group of children treated, equivalent to 104 patients for six months, the only child possibly suffering from a recurrence was aged eleven years, and was readmitted to hospital complaining of anorexia and lassitude four months after the onset of her previous attack and three months after commencing sulphanilamide treatment. Her hæmoglobin value was 89%, her temperature and sleeping pulse remained normal, her blood sedimentation rate estimated by Cutler's method was four millimetres per hour, and her cardiac condition did not alter appreciably. The blood sulphanilamide level was 1·7 milligrammes per 100 cubic centimetres. It seems that the diagnosis of "recurrent rheumatic fever" was doubtful.

The control group consisted of an average number of 470 children who were on the roll of the rheumatic clinic, and who were not taking sulphanilamide during the same six months. However, 100 of these children did not attend once during these months, and of those who did attend, a

[1] Read at a meeting of the Melbourne Pædiatric Society on March 14, 1945.

small proportion had not had an attack of rheumatic fever during the preceding five years. Of the 470 children, thirty definitely and three possibly suffered a recurrence of the disease during these six months; this indicates a recurrence rate of 13·5% to 15% per year, estimated in this case from the summer season (August to February).

American literature[2] suggests that the occurrence of acute rheumatic fever in the summer is rare, and the practice in America appears to be to stop prophylactic sulphanilamide therapy during the warm months. Similar circumstances do not exist in Melbourne, though probably our incidence of rheumatic relapse is much higher during the winter than during the summer.

Blood Sulphanilamide Level.

The concentration of sulphanilamide in whole blood was estimated by the method of Marshall in a number of cases. In twelve of these the level ranged from 1·3 to 3·2 milligrammes per 100 cubic centimetres, and this was considered satisfactory. In the blood of the remaining few children tested, no drug could be found, and it was presumed that the patients were not taking the drug. This view was supported by the detection of a rise in the blood sulphanilamide concentration to a satisfactory level when the same children were admitted to hospital and the drug was given in the above-mentioned dosages.

Toxic Effects of the Drug.

In the large majority of cases, the toxic effects of sulphanilamide were not serious.

The most frequent complaints were of macular skin rashes. These occurred in thirteen children; of these only three were permanently prevented from continuing to take the drug. In general, the rash was morbilliform; it appeared on the extremities, usually the legs, and later extended to the trunk.

Nausea and vomiting affected some children in the first few days of treatment, and finally prevented three patients from continuing.

Persistent cyanosis in two cases, and leucopenia below 5,000 cells per cubic millimetre in two more cases, were reasons for stopping therapy. The leucopenia quickly disappeared without treatment.

Two children failed to attend for their appointment, even when requested by letter, and no further efforts were made to seek their cooperation.

Altogether, thirteen of the children who commenced taking sulphanilamide discontinued the drug. One child was transferred to another hospital.

It was surprising how rapidly and completely the minor inconveniences mentioned above disappeared after the affected patients had continued to take the drug for two or three weeks.

Further Observations.

At least fourteen children taking sulphanilamide were referred for tonsillectomy, and a similar number for dental extraction.

Claims have been made and refuted[3] that tonsillectomy prevents rheumatic recurrences; our object in advising tonsillectomy was to remove what was considered, in each case, a hopelessly diseased and therefore a useless and dangerous organ. It was the general impression gained by those of us who removed tonsils from these children that hæmorrhage was more profuse and of longer duration than in the case of children not taking sulphanilamide. Further, the frequency of epistaxis was judged to be rather greater among those children given sulphanilamide than among the same children before they began prophylactic treatment. Routine tests estimated bleeding and clotting times to be normal.

Tonsils removed from ten children taking sulphanilamide were investigated for their content of hæmolytic streptococci. Tonsils from four of these patients yielded β-hæmolytic streptococci group A; the other six yielded none.

The views of Collis[5] are interesting in this regard; he has found streptococci in the depths of tonsils from patients who had died of rheumatic fever. Examination of a small number of routine throat swabs taken from children being treated in our clinic failed to reveal any group A hæmolytic streptococci.

Many times we were confronted by children in their first two months of sulphanilamide prophylaxis, who complained that they were suffering more than previously from pain in muscular and fibrous tissue and occasionally in joints. Such pain was described as lasting a few minutes only and was generally mild; it was not associated with any general symptoms and disappeared as quickly as it came.

It is clearly realized that chemotherapy is not the final answer to the problem of rheumatic fever; the disease is largely, though not entirely, one of poverty and malnutrition, associated with social maladjustment and parental inability or neglect; but it is hoped that prophylactic measures, such as those described above, may protect these unfortunate children until more fundamental adjustments can be made.

Summary.

During the summer months, from August, 1944, to February, 1945, prophylactic sulphanilamide therapy was commenced among 160 children convalescent from rheumatic fever. This group was estimated to be equivalent to 104 patients each observed for six months. Of these, one child suffered from a doubtful recurrence of the disease. An average of 470 children not taking the drug formed a control series during the same time, and of these thirty definitely suffered from recurrent rheumatic fever, and three possibly so; this is equivalent to a recurrence rate of 13·5% to 15% per year. The toxic effects of the drug were minimal. Aspects of the treatment are briefly discussed, and several relevant observations are recorded.

Acknowledgements.

I should like to express my thanks to Dr. H. Laurence Stokes, director of the rheumatic heart clinic, without whose help, encouragement and advice the establishment of this routine work would not have been possible, and also to Dr. G. E. M. Scott, Dr. H. J. Sinn and Squadron Leader D. Hamilton for their cooperation.

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INJURIES PRODUCED BY PLANTS IN TROPICAL QUEENSLAND.

By H. FLECKER, M.B., Ch.M., F.R.C.S., F.F.R., D.R.,
Cairns.

IN a series of four papers Professor J. B. Cleland has collected a numerous group of injuries produced in Australia by various plants. The following embrace some of those reported to the Registry of Injuries Produced by Plants and Animals in Tropical Queensland, established some years ago, and include some not so far listed by Professor Cleland.

Family Euphorbiaceæ.

Jatropha multifida, although a native of Central America and Brazil, has been called "French physic nut".

M., aged thirty-six years, reported by Dr. D. P. O'Brien, of Rockhampton, chewed the nut on April 14, 1936. It tasted sweet and pleasant and he swallowed only one nut. Agonizing pain began half an hour later, with constant vomiting, diarrhœa, cramps, numbness and paralysis in the legs and arms. The patient felt as though he was dying and collapsed; he was pulseless with cold sweats, numbness and tingling in the arms and legs. This lasted for twenty-four hours and he recovered in a week.

The plant is commonly cultivated as an ornamental bush and has bright red flowers. Some notes on this and other species of Jatropha are to be found in Byam and Archibald's "Practice of Medicine in the Tropics", Volume I, 1921, page 780.

Family Hippocrateaceæ.

Siphonodon pendulum is a native of North Queensland and its fruit resembles somewhat in appearance a very hard mandarin orange with a smooth surface. The following are reported by the Reverend D. A. Brown from Weipa Mission Station, Gulf of Carpentaria, 150 miles south of Thursday Island.

Three aboriginal girls, all aged eight years, swallowed fruit on July 21, 1938. This was followed by severe pains and convulsions. One who ate three fruits died, but the others, who had taken one each, recovered, and the same evening appeared to be well.

Family Anacardiaceæ.

Semecarpus australiensis is the "marking nut" tree of F. M. Bailey, but commonly known in North Queensland as tar tree, owing to the black tarry sap beneath the bark.

R.B., a male, aged twenty-three years, was one of two men working on a large tree on September 24, 1936, for two hours grubbing roots with adze, ax and shovel. He did not handle any part of the trunk. No deleterious effects were noted for two days, when a very itchy rash appeared, attributed to the splashing of the juice by the ax. A papular rash situated immediately above the elbow was still present at the time of the examination on October 8, 1936. Isolated patches were also present on the right forearm. The victim, who wore trousers clipped at the ankle, had a dermatitis on the posterior aspect of the right thigh, the left inguinal region, scrotum and penis, which he attributed to the green tree ant (*Oecophylla virescens*) entering above from below the belt causing irritation and scratching. His mate did not suffer from any rash.

D.H., a male, aged fourteen years, was climbing a tree on December 22, 1940, when a branch broke. Small papules appeared about the chin and sides of his face and front of the neck, but there was no rash on his hands. These were attended with much irritation and still persisted when examined eight days later.

The "tar tree" is a very large, handsome tree, native to North Queensland, with deep green leaves providing an abundant shade and formerly was plentiful about Cairns, but owing to its unmerited reputation as a source of irritation, its numbers in recent years have unfortunately been greatly reduced.

Family Cunoniaceæ.

Davidsonia pruriens (Davidsonian plum) is native to North Queensland; its fruit resembles a large plum of a deep purple colour and is particularly sour to the taste. Nevertheless some children will eat it. It contains two small flattened seeds within the fruit. The following case is reported by Dr. H. S. Harper, of Mossman.

J.H., a female, aged ten years, was admitted on May 18, 1941, to the Mossman Hospital with epigastric pain and vomiting with a temperature of 99·2° F. Dr. Harper was unable to make a diagnosis for two days, when the patient vomited some grey fluid which contained the thin flattened seed, afterwards recognized as of *Davidsonia pruriens*. Prompt recovery occurred after the vomiting.

Family Compositæ.

Acanthospermum hispidum (star burr) is a common introduced weed.

J.H.W., aged sixty years, complained of pain and tenderness over the right *tendo Achillis* caused by the burr of this weed, which was forced in through the skin by the boot. The lesion took six weeks to heal.

Family Solanaceæ.

Capsicum fastigiatum (chilly) occurs in North Queensland. In April, 1943, a report appeared in the *Cairns Post* that a child suffering from "bad burns on the face and hands" had been attended by a member of the Queensland Ambulance and Transport Brigade after picking and eating chillies, with the note that these bushes "grow profusely around Cairns, and wherever one is noted it should be eradicated". However, it appears that the victim was not attended by a medical man, and it seems that this isolated record scarcely justifies the extermination of this common bush in North Queensland. The effect of eating the fruit of this useful and ornamental plant is well known and most children will surely avoid it after the first taste.

Family Araceæ.

Dieffenbachia picta is a small introduced ornamental plant grown in pots or gardens and is easily recognized by the white splotches on the back of the green leaves.

D.F., a male, aged two years, on April 8, 1936, was seen pulling at the plant in the garden. He vomited once, complaining of soreness of the mouth and swollen lips, and was holding his tongue in his hand. When seen two and a half hours later he was apparently well.

V.M., a female, aged fifteen months, on October 24, 1936, chewed the stem of the plant, while still growing in the pot; she left teeth marks on the stem. She had mucous secretion from the mouth and cried for half an hour. She was well an hour later.

D., a female, aged twenty-one months, picked a leaf and put it into her mouth. This was followed by severe pain in the mucous membranes of the lips and cheeks and she cried for a considerable period. The mucous membrane of the cheek was swollen. She vomited once. Tingling pain came on three minutes afterwards and lasted for forty-five minutes.

There was also a history of other children being also slightly burnt, and a calf a few months old was badly affected.

Family Palmaceæ.

Caryota urens is a palm introduced and cultivated for ornamental purposes. The fruit is of a deep purple colour and of about the size of cherries, borne on long spikes.

L.W., a municipal gardener, on January 5, 1940, when carrying the ripe fruit of this palm on both forearms immediately felt an acute stinging pain on both forearms at the points of contact. The pain lasted five hours.

Reports of Cases.

INJURY TO THE POPLITEAL ARTERY.

By J. M. YEATES,
Major, Australian Army Medical Corps.

(From an Australian Casualty Clearing Station.)

"AT a surgical conference held in Cairo in July, 1943, there were very few surgeons who could remember an instance in which ligature of the popliteal artery in the forward areas had not been followed by gangrene."(1)

"The popliteal vessels, like the femoral, have an evil reputation among military surgeons for the high incidence of gangrene which follows their occlusion. For this reason conservative methods are indicated wherever possible, and ligation should be employed only as a last resort."(2)

These two quotations serve admirably to show the consensus of informed surgical opinion regarding the danger of gangrene attending ligature of the popliteal artery. The few references available to the writer at the moment would seem to show that the gangrene rate varies from 30% to 50%.

Clinical Record.

Private "D", aged twenty-eight years, was admitted to this unit at 11 a.m. on November 16, 1944. In his own words, he had been "hit with a bit of shrapnel" at 10 a.m. the previous day. The delay in evacuation was due to the apparent minor nature of the wound. There were no symptoms, his only disability being a slight limp.

Examination revealed a small punctured wound in the midline of the left popliteal region, some four inches above the joint line. No swelling was present in this area, nor were there any other physical signs of note. The leg and foot were of the same colour and temperature as those of the unaffected limb. Neither *dorsalis pedis* artery was palpable. X-ray examination revealed a complete bullet, lying almost horizontally, near the postero-medial border of the femur, some three and a half inches above the lower end of this bone. The apex of the bullet was directed forwards. It was clearly very close to the popliteal vessels. In view of this finding a further clinical examination was performed for signs of circulatory disturbance, again without result. There was no evidence of injury to the tibial nerve.

Operation was performed under anæsthesia by ether introduced by the endotracheal route at 2.30 p.m. on November 16. The patient was placed in the prone position. An Esmarch's tourniquet was applied to the upper third of the thigh. A three-inch axial incision was made, centring about the wound. When the deep fascia was incised, no blood escaped; but the wide belly of the semimembranosus muscle prolapsed. The track of the bullet was clearly seen in the muscle, which was retracted medially. After a slight quantity of fresh blood clot had been mopped up, the rear end of the bullet was just visible in the depth of the wound. Offering a little resistance, the bullet was then removed. As the cavity from which it had been extracted was very deep and inaccessible, the exact anatomical details were not obvious. For fear of injury to the popliteal vein, no further dissection was deemed advisable or really necessary.

The tourniquet was now released. No bleeding occurred. The wound was insufflated with penicillin powder, and "Vaseline" gauze was applied. The track in the muscle was not excised, because of its clean appearance, and because of the minor extent of the damage to the fibres. No sutures were inserted. The leg was then lifted in readiness to apply the bandage, when a sudden gush of blood was seen. As this blood appeared rather dark in colour and gushed rather than spurted, it was considered to be coming from a tear in the popliteal vein. For this reason the leg was elevated, and the wound was packed with gauze. This controlled the bleeding until further movement of the leg caused furious hæmorrhage. It was now quite clear that the popliteal artery had indeed been injured. The tourniquet was immediately reapplied, the incision was enlarged, and a thorough exposure of the popliteal vessels was effected.

At this level, the popliteal vein lies almost directly posterior to the artery, a little to the outer side. For this reason it was surprising to find the vein uninjured. The popliteal artery was found to have a tear involving quite three-quarters of its circumference. Proximal and distal ligation was performed. The injured segment was excised completely. The vein was not ligated. When the dressings were applied and the tourniquet was removed, the left foot was at once seen to be slightly pinker in colour than the right. A Thomas splint was then applied.

For the next few hours the colour of the foot was watched rather anxiously. However, at no time during the next few days was there the slightest suggestion of circulatory failure. Sensation and movement of the foot were normal.

The patient's temperature rose to 100° F. on November 17, but soon subsided under the influence of a course of sulphamerazine. Secondary suture was performed under local anæsthesia on November 22, the wound being free from signs of infection. Convalescence was delayed because of difficulty in obtaining full extension of the knee joint owing to the longitudinal incision. The patient was finally discharged to his unit for full duty on January 2, 1945.

Comment.

In the compilation of statistics for gangrene following popliteal ligation, it is suggested that the exact anatomical level of the lesion be stated. In the few references obtainable in this area this fact never received notice.

The popliteal artery is actually some seven or eight inches long. In this case the lesion was very high, being about one and a half inches distal to the *hiatus tendineus*, and proximal to all five genicular arteries. This is possibly the reason why the collateral circulation was so rapidly and adequately established. It is also conceivable that some blood was able to flow past the tip of the bullet during the period prior to operation, although this seems improbable.

The point is, that if the literature shows that all high ligations are attended by equally favourable results, then the surgeon need have no qualms about applying this positive form of treatment in every such case, reserving arterial suture, with its greater technical difficulties and less successful results, for more distal lesions.

In short, is there a crucial point in the popliteal artery analogous to the "critical point" of Sudeck in the inferior mesenteric artery?

Acknowledgement.

This report is published with the permission of Major-General S. R. Burston, Director-General of Medical Services.

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Reviews.

AN ENGLISH TRANSLATION OF GALEN.

NOT infrequently the comment is heard that it is difficult to procure an English translation of the works of the great medical dictator, Claudius Galen, who practised his profession laboriously and wrote voluminously throughout the latter half of the second century of the Christian era. When Arthur J. Brock published in 1929 his book of selected passages from some of the outstanding Greek medical writers, he said that his translation of "The Natural Faculties", which appeared in the Loeb Classical Library series of 1916, was probably the first English rendering ever published of any work of Galen. It is, therefore, all the more gratifying that an English translation of Galen's treatise "On Medical Experience" has at last come to light, together with the Arabic text, which was discovered by Dr. H. Ritter in a library at Constantinople only fourteen years ago.[1] For this first edition of the Arabic version we are indebted to Dr. R. Walzer, of Oriel College, Oxford, whose ripe scholarship has given us a translation that makes most agreeable reading.

This interesting volume, handsomely produced by the Oxford University Press, takes us back to the very foundations upon which the medical science of our own time has been slowly and haltingly built up over a long stretch of centuries. From Galen's own words we learn of the difficulties experienced by ancient physicians in their efforts to find a logical explanation for the workings of the body in health and disease. Hence the gradual emergence of different medical sects or schools of thought in Græco-Roman times; each promulgated freely its own particular theory of human physiology and pathology and violently contested the correctness of its assertions with its rivals in the profession. In his treatise Galen tells us about the Dogmatists, who were the acknowledged followers of the great Hippocrates, and so made a theoretic system out of the doctrine of the humours and qualities; the Empirics, who went to the other extreme and refused to depend upon anatomy and physiology, relying on clinical observation and their own experience. On the other hand, the Methodists held that all disease was due to an improper arrangement of the particles and pores of the tissues; and in the opening passages of his book, Galen, who rather prided himself on belonging to the Eclectics, reveals his contempt for the chief exponent of Methodism, Æsclepiades of Bithynia, a Greek physician who practised in Rome about 60 B.C.

Galen maintained his reputation as the guiding star of all medical philosophers for over thirteen hundred years; and his works were carried on far into the Renaissance period by virtue of the fact that the Mohammedan invaders set great store by the ancient manuscripts of the Greek writers, and these were duly translated by able scholars who were deeply respected and amply rewarded for their work by the Islamic rulers. Galen's book "On Medical Experience" was first translated from the Greek into Syriac by the Nestorian physician, Hunain ibn Ishaq, in the ninth century, and then his nephew later translated the treatise from Syriac into Arabic. Hunain, considered the most profound of the Oriental scholars, achieved the colossal task of translating into Arabic all the *Corpus Hippocratica*, all the writings of Orabasius, Paul of Ægina, and the text of Dioscorides on pharmacology; and now his version of Galen's book, the original Greek text of which has been lost, is given to us as the earliest version at present known. Hence the value of this splendid publication to the student of the history of medicine and to all who seek insight from the past for a better understanding of their art.

[1] "Galen on Medical Experience", First Edition of the Arabic Version with English Translation and Notes, by R. Walzer, 1944. London: Oxford University Press. 9½" × 7", pp. 174. Price: 12s. 6d.

The Medical Journal of Australia

SATURDAY, JUNE 23, 1945.

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SCIENTIFIC RESEARCH AND THE FUTURE OF MAN.

In one of his later essays Professor Walter Murdoch describes the "strange behaviour of a gramophone". He imagines what he hears in a dream from an enraged gramophone, a talking machine, acting as the mouthpiece of all machines. Machines, it tells him, were created "to save labour, to rescue men and women from drudgery, to do the hard physical work of the world". Man has arranged, "with a kind of insane ingenuity", that all the blessings which machines were meant to bring are turned into curses. Instead of leisure and plenty, only unemployment, poverty and want have been achieved. The machine ends by saying to him: "Until you wake up and make the necessary changes in what you call your 'system' so as to allow us to usher in an era of abundance and of leisure, we shall kill as many of you as we can—for we think that such a race as yours deserves to be obliterated—and then burst our boilers and crack our pistons and generally scrap ourselves." During the last few years machines and engines of war have done their worst, and if the race has not been obliterated, civilization has been brought to the brink of destruction. There is understanding behind the statement sometimes made (though those who make it do not intend to be taken literally) that the only way in which to check the rising horror of wars is to kill off all scientists. Bertrand Russell put this another way when he wrote: "The new powers that science has given to man can only be wielded safely by those who, whether through the study of history or through their own experience of life, have acquired some reverence for human feelings and some tenderness towards the emotions that give colour to the daily existence of men and women." Man is naturally inquisitive. He is not content to live from day to day an unintelligent existence, asking for no more than food and clothing. He wants to know "why" and also he wants to find ways and means of wresting from Nature some of her secrets that he may make life more agreeable by changing his environment where he finds that it does not favour his well-being and progress. But, as we have seen, in the application of his knowledge man has turned aside from wisdom.

Many people think that the disordered and partially dismembered world of today is a forerunner of still more dreadful states of upheaval, largely because scientific discovery yet to be made, instead of being harnessed in the service of man in peace and progress, will be turned to his destruction. Inquiry into what should be done about this state of affairs will elicit different opinions. One person will declare that no more scientific discoveries should be permitted for a stated length of time, perhaps accompanying the remark with the suggestion already mentioned that all scientists should be segregated, if not destroyed. Another will hold that only research likely to benefit man should be undertaken. (These people do not say who is to enforce these conditions.) Again, the view will be expressed that every new discovery should be made common property, with the possible rider that every nation should be party to an international agreement not to use certain specified processes in the preparation of armament. Such measures, could they be undertaken, would be either ineffective or impracticable. Taking the last first, we all know from experience in our own days that international agreements are not reliable safeguards, that a plausible excuse can when necessary be produced for their infringement, or that an excuse, plausible or not, may not even be considered. Again, at the inception of research no question of its possible benefit to man may arise. Many an isolated discovery has been dovetailed with one or more others to produce a new instrument for the use of man. Others intended to benefit man have by his perversity or evil design been, as declared by Professor Murdoch's gramophone, turned to curses. The view that scientific research of all kinds should be suspended for a period, though impracticable, and even if it could be arranged never likely to be adopted, probably has the right idea behind it—that in the latent period man might have time to prepare his mind to receive new data from science, to adjust his ideas to them, and to apply them in accordance with his newly acquired sense of values. The truth of the matter is that education has not kept pace with advance in knowledge, and that as a consequence man's acquisitive instincts with their concomitants of hatred and violence have possessed him and threaten him with destruction.

To the question of whether man in his present volcanic state of unrest is beyond hope, there is only one answer. To say that he is not redeemable would be pessimism of the worst order. It would deny the inherent qualities of goodness of every man, smothered under the self-wrought ruins of his character though they may be. Man can be educated, "led out" to act both individually and in his larger national groupings as an intelligent and sentient being with understanding and with purpose based on his better qualities. Plato wrote that it was not the life of knowledge, not even if it included all the sciences, that created happiness and well-being, but a single branch of knowledge—the science of good and evil. These words are quoted by Sir Richard Livingstone in the opening chapter of a small book entitled "Education for a World Adrift", published in 1943. This work, which deals with the present

world problem in a clear and unequivocal way, should be widely read. Livingstone holds that the great task of the present generation is to find a principle that will rule life. In order to do this we must realize that we are in the midst of two revolutions, the one social, economic and political, the other spiritual, "the weakening or the dissolution of the traditions and beliefs which for many centuries have ruled Western civilisation and held men together". We all live in two worlds—"in the everyday world which changes in each age with the progress of material civilisation; and (whether we give it that name or not) in a spiritual world, without which Science could not believe in Truth, or Religion in God, or Good itself have any meaning". The efficiency of a country in Livingstone's opinion will depend on its technical and vocational education, its cohesion and duration largely on its social or political education. The quality of its civilization depends on its standards, its sense of values, its idea of what is first rate and what is not. It is fatal to omit this spiritual aspect because its absence is like an insidious disease which may advance to an incurable stage before its existence is recognized. Livingstone makes a good point when he writes that education is incomplete unless it gives to everybody an intellectual attitude to life and a philosophy of life. He defines the right intellectual attitude to life as threefold—to find the world and life intensely interesting, to wish to see them as they are and to feel that truth is both permanent and beautiful. The right intellectual attitude to life is already, he thinks, a partial philosophy of it. This philosophy is complete if it is extended to cover goodness, truth and beauty, and if goodness is defined to cover love, justice, courage, self-mastery, mercy and liberty. If we suppose that every citizen is an educated man in the fashion here described as complete, he will instinctively take notice of those who live around him, he will take his proper place as a citizen. Citizenship, Livingstone states, involves all man's actions that touch his fellow citizens and affect the health and well-being of the State—"it is almost co-extensive with his duty to his neighbour". Citizenship must be taught. It may be taught in the way that instruction is given in schools, but this, though desirable, is not nearly enough. The best way to learn citizenship is to practise it. It should be possible to "give everyone a chance of thinking about life when he is facing it and about its problems when he has to solve them". Citizenship is "conduct not theory, action not knowledge". It is possible to learn all that is to be known in books on the social sciences without being a good citizen. By practising the qualities of citizenship we may develop a community sense, and the art and virtue of living in a community is, Livingstone tells us, the most important element in citizenship. To live the community life calls for a display of moral qualities associated with the idea of service, and Livingstone quotes Whitehead's statement that moral education is impossible without the habitual vision of greatness. To this he adds Aristotle's dictum that men acquire virtues, not by knowing what they are, nor by talking about them, nor by admiring and praising them, but by practising them. A great deal more might be written about citizenship, its acquisition and its proper use, but we must return to our original discussion. In education lies hope for the future. It must be undertaken by individuals and by organizations. Matters such as this are always left to a few—acting either as unattached individuals or as individual members of an organization. A public opinion must be created. It can be done. It must be done. The aim must be to build communities comprising individuals who have ideals of citizenship, not only of national areas, but of the world. The aim cannot be too high. Without striving to achieve such an aim man will not be able to inhabit the earth without fear of diabolical inventions that will destroy him; but if he does, he may make a close approach to his goal and to the fulfilment of his highest destiny.

Current Comment.

PRIMARY BRONCHIOGENIC CARCINOMA OF THE LUNG.

FROM time to time reference has been made in these columns to the commonly held view that cancer of the lung is of much more frequent occurrence than it used to be. Certain authors hold the view that the condition has shown both a relative and an absolute increase; others are more cautious and refuse to admit that satisfactory evidence of a real increase has been produced. Last October observations of P. E. Steiner were recorded. Steiner is one of those who will not admit that a real increase has been proved. His observations showed that the general increase in lung carcinoma, estimated in five-year periods, was striking, but that the increase when controlled by comparison with increases in other forms of carcinoma, did not appear to be excessive. An interesting study on bronchiogenic carcinoma has been published by A. Ochsner, J. L. Dixon and M. De Bakey,[1] who state that the incidence of pulmonary malignant disease is increasing both relatively and absolutely. The incidence and the ætiology are important from the purely clinical point of view, and it is to this aspect of Ochsner, Dixon and De Bakey's paper that we direct attention. These three observers are among those who regard tobacco smoking, particularly in the form of cigarettes, as one of the chief causes of cancer of the lung. Others have tried to incriminate tuberculosis or influenza. Incidentally, it may be remarked that if cigarette smoking is the chief cause, the predominance of male patients over females with cancer of the lung should before long disappear. Ochsner and his co-workers can find no role for outdoor occupations in the causation of the condition. They point out that bronchiogenic cancer is relatively slow growing and remains localized for a considerable period of time. In these days, when pneumonectomy has become the established treatment for malignant disease of the lung, it is important that diagnosis shall be made early. From this point of view the question of cigarette smoking and cough are important. Delay may be caused by the assumption that a chronic cough is due to the fumes of tobacco when really it is the result of early carcinoma. Unfortunately there are no characteristic signs and symptoms of bronchiogenic carcinoma. As Ochsner, Dixon and De Bakey point out, it can simulate almost anything; the onset is usually insidious and the symptoms are likely to be attributed to some other cause. Many of their patients dated their illnesses as beginning with an attack of influenza or some infection of the lower part of the respiratory tract. R. C. Brock, writing in the *British Medical Journal* of August 28, 1943, stated that by far the most important group of symptoms arose from the infection that sooner or later followed upon bronchial obstruction. Of 52 patients submitted to pneumonectomy by Ochsner, Dixon and De Bakey, 47 suffered from cough, 33 had loss of weight, 29 had had previous respiratory infections, 29 had had hæmoptysis, 25 dyspnœa and 17 pain in the chest.

[1] *Diseases of the Chest*, March-April, 1945.

Clearly the findings will vary with the site of the tumour in the lung—occlusion of a bronchus will cause atelectasis. Brock holds that general deterioration of health, loss of weight and loss of strength are not signs of carcinoma of the lung, but of approaching death. The most reliable method of making a diagnosis is by bronchoscopy and, of primary importance, a constant realization of the possibility of the existence of cancer. The value of bronchoscopy has recently been shown by N. R. Pillsbury and J. D. Wassersug,[1] who found that twelve patients with bronchiogenic carcinoma had been admitted to a tuberculosis hospital.

A final point that should be mentioned is the duration of the condition. Ochsner, Dixon and De Bakey quote many authors who hold that the disease progresses rapidly; this has not been their experience. They believe that most primary bronchiogenic carcinomata are relatively slow-growing and can exist for a long period of time before symptoms are produced. This is a statement that should be remembered by clinicians when they contemplate a diagnosis of malignant disease of the lung.

MASS MINIATURE RADIOGRAPHY AS A PRACTICAL MEASURE.

THE subject of mass miniature radiography has been dealt with extensively in the literature, and most current communications now concern the practical application of the method to various branches of the community. Of these we are now more concerned with the civilian, and must plan for the future. A very valuable brochure has been recently published in London by the Medical Research Council dealing with the details of organization necessary for the mass radiography of large groups.[2] This excellent manual sets forth with full detail the particulars of equipment suitable for the use of a mobile team, the personnel required, the clerical routines, the actual running of the organization and the principles and practice of diagnosis of thoracic disease as met with in the survey. There are a number of very clear and useful photographs illustrating methods and routines. Considerable service experience has been gained in Australia in miniature radiography on a large scale, but it may be pointed out that difficulties such as the employment and payment of the team hardly arise in the services, nor is there any need for "selling the investigation" to the members of the public. Some local experience has also accumulated concerning the examination of large groups of volunteers. In Adelaide most valuable work has been done in the past, as the records of this journal show, and H. K. Fry's recent survey of the results of Mantoux tests has supplemented this radiographic work and added to the valuable immunological inquiries of D. Anderson and others. This preliminary work among civilians must be of great help in planning further surveys. The question of attracting volunteers to undergo investigation is of itself most important, as the Medical Research Council's team point out. They stress the need for correct publicity; where large groups of industrial workers in any given industry are being examined the cooperation of the works medical officer is vital for success. He must understand the need for professional secrecy, and agree that he will learn the results of individual tests only from the persons concerned, for to suggest compulsion is to invite opposition. The ground for a survey must be prepared beforehand, and a steady flow of volunteers assured. Then accommodation for the team and the apparatus must be found and prepared. This involves many considerations, such as current supply, office space, water, arrangements for protection of the staff and so forth. The technical details of the equipment need not be dealt with here, except to remark in passing that a highly luminous screen (the Levy-West mark 39) and a British camera with a new lens of two-inch focal length and f./1·5 aperture were used with success. Governments and other public bodies interested in these surveys must realize from the start that, important though costs are, efficiency and even accuracy will suffer unless the team is adequate in number and capacity. A permanent team of eight dealt with the actual radiographic examinations. A part-time radiologist was employed, and a medical director, preferably full time, with a personal clerk. The director dealt with people before and after the examination and initiated any necessary follow-up of doubtful cases. He also carried out certain diagnostic procedures. In addition to this team a certain base staff is also necessary.

Such efficiency was achieved that equipment could be installed in a new place and a test run made within a few hours. Full descriptions are given in this publication of the office methods adopted; these were of particular interest in those cases in which for any technical or diagnostic reason one of the volunteers was required to return for a full-size film of the chest. Every effort was made to follow up the doubtful cases, and here again cooperation was invited and compulsion never hinted.

When a diagnosis of tuberculosis was made the local tuberculosis officer was informed, the large films and notes were sent to him either through the family medical attendant or direct, and an appointment was arranged. The methods of diagnosis are discussed briefly but clearly. Physical examination, estimation of the blood sedimentation rate, examination of the sputum both direct and by culture methods, were carried ont, and in selected cases culture was made from a direct laryngeal swab. The results of the survey of 23,000 persons are given in detail. As an example, of a factory group of 5,200 available employees, 90% attended for examination; of these 5·5% were recalled for large films, and of this fraction 2·5% were further medically investigated. Tuberculosis of significant type and degree was disclosed in 1·5% of the total, and treatment found to be necessary in 0·4%. In addition a number of non-tuberculous lesions were discovered.

Accounts of several of the bacteriological surveys carried out in suspect cases have been published from time to time in this journal; the Medical Research Council's team refer to the excellent work of Brooks in the Royal Navy, Trail and Clive in the Royal Air Force, and Bigger in the British Army, and to that of Webster on Australian army material. The precision and value of such work cannot be over-valued, and any future surveys of civilians must provide such facilities. In the present inquiry, among 318 cases bacteriologically investigated evidence of active tuberculosis was proved in 73. This brochure may be commended to the attention of all those interested in this important branch of preventive medicine. Its practical details should be of great help to future workers in this field, and its brief epidemiological sections indicate that much fruitful work can be done along these lines in future surveys.

Here in Australia we have most of the requirements for such surveys ready for future work. Already in some centres miniature radiography is freely available to the public at low cost. There are numbers of clinical, radiological and pathological experts available who have amassed great experience already. Before the war it was fortunate that interest and research in miniature radiography were alive and active, thus enabling us to make a flying start. But once more it is essential to look well ahead, for there are several points at which plans for mass surveys may break down. Equipment we can obtain, but can we be sure that the governing bodies concerned will realize the necessity for an adequate staff, both lay and technical? In order to make accurate diagnosis in the suspect cases time, skill and money are required. And once the condition is diagnosed, will the subjects of tuberculosis find facilities for modern treatment available? Finally it is to be earnestly hoped that encouragement will be given to more medical men and women to take up tuberculosis work whether in private or institutional or preventive practice. It requires special aptitude and training, and must not be regarded merely as a side-line,

[1] *The New England Journal of Medicine*, March 8, 1945.

[2] "Mass Miniature Radiography of Civilians for the Detection of Pulmonary Tuberculosis", by Kathleen C. Clark, P. D'Arcy Hart, Peter Kerley and Brian C. Thompson; Medical Research Council, Special Report Series, Number 251; 1945. London: His Majesty's Stationery Office. 9¼" × 6", pp. 139. Price: 3s. 0d. net.

Abstracts from Medical Literature.

MEDICINE.

The Treatment of Scarlet Fever.

MAX J. FOX AND NORMAN F. GORDON (*Archives of Internal Medicine*, July, 1944) discuss the treatment of scarlet fever, review the literature, and analyse the results of therapy in a series of 2,000 cases. Sulphonamide compounds were found to have their chief value in the treatment of certain complications, but were of no value in the management of the toxic phase or type of scarlet fever. Commercial antitoxin, prepared with horse serum, combats the toxic phase of the disease, but introduces the danger of foreign protein reactions. Pooled human convalescent serum produces rapid clinical response when administered to patients with scarlet fever and offers the best means of therapy. The notable beneficial effects of its use include prompt subsidence of fever, alleviation of signs and symptoms, avoidance or improvement of complications, shortened period of hospitalization and lower mortality rate. Smaller doses of convalescent serum than have been previously used were found to be effective.

Diabetes Insipidus.

GEORGE M. JONES (*Archives of Internal Medicine*, August, 1944) discusses *diabetes insipidus* and presents a report of the important features based upon a study of 42 cases of the disease. The historical and experimental background of the disease is reviewed, and the ætiology and pathology are described. *Diabetes insipidus* is a symptom complex produced by injury to the supraoptico-hypophyseal tract, and is not a specific ætiological entity. Thorough and repeated examinations should always be made to determine the ætiological factors. Urine concentration tests indicate that patients with *diabetes insipidus*, who are receiving a restricted amount of fluid, continue to secrete urine of low specific gravity, with resultant loss of body weight. In any case in which such a response is obtained, the diagnosis of organic damage to the supraoptico-hypophyseal tract should be made. The ideal therapy should be directed toward the ætiological factor, if determined. In the author's series of 42 patients, there were 34 patients in whom the ætiological factor was determined. In some cases of neoplasm of the hypothalamus and pituitary, and of Hand-Schüller-Christian disease, a good response to X-ray irradiation is obtained, and antisyphilitic therapy corrects the *diabetes insipidus* resulting from syphilis. *Diabetes insipidus* is not infrequently the first symptom of neoplasm, and X-ray irradiation of the hypothalamico-hypophyseal region may well be worth while when the cause of the diabetes is undetermined. The various methods of administration of posterior pituitary gland extract as replacement therapy in the absence of the antidiuretic principle, are discussed and analysed in relation to the results achieved in the treatment of the author's patients, and the intramuscular injection of pitressin tannate in oil is regarded as the most desirable at present. Intranasal application of pitressin in jelly was found to be the least satisfactory of the methods tried. The use of a diet with a low salt content as an adjunct to other therapy is considered to be worth a trial in any case of this disease. Thyreoidectomy should not be performed for *diabetes insipidus* unless there are other specific indications for this procedure.

Brain Abscess following Thoracic Disease.

J. L. COLLIS (*The Journal of Thoracic Surgery*, December, 1944) has sought a mechanical explanation of the occurrence of cerebral abscess after suppurative thoracic disease and one which would include a route for the transference of infected material to the brain. The known facts concerning the disease fall into two groups. First, there are negative facts which by virtue of their equal distribution do not throw any light on the subject of the ætiology, such as age incidence, side of the brain abscess, site of the brain abscess, the infecting organism, the number of abscesses present and the site of the primary disease in the chest. Secondly, there are apparently significant facts such as the greater frequency in the male sex; the greater rapidity with which the disease reaches a fatal termination compared with cerebral abscess due to other causes; the essential similarity of all aspects of cerebral abscesses in this group whether they follow lung abscess, empyema or bronchiectasis; the fact that the cerebral complication may follow disease in the lung or disease in the pleura; the fact that only chronic disease or disease that has become chronic causes cerebral abscess; the increased danger after surgical intervention; and the relative absence of abscesses in other parts of the body in association with the cerebral involvement. The recent rediscovery of the very free communications which exist between the spinal veins and sinuses of the *dura mater* above and the intercostal and lumbar veins below seemed to supply the very explanation sought, especially as the spinal veins are valveless and blood can flow through them readily in either direction. The author injected opaque material into an intercostal vein in cadavers and found by subsequent X-ray examination that it flowed into the vertebral venous plexus and up and down the spine and into the skull with remarkable freedom. He propounds the theory that infected emboli entering the intercostal veins are the cause of cerebral abscesses: should they be carried to the azygos veins and not to the spinal veins, they may be "caught up in the pulmonary bed without ill effect". Operative interference obviously increases the chances of the occurrence of such septic embolism. The special importance of chronic conditions is due to the fact that it takes considerable time for pleural adhesions to become vascularized, so that a thrombotic process can spread across them from the lungs to the intercostal veins.

Carbon Dioxide as an Expectorant.

A. L. BANYAI AND A. V. CADDEN (*The British Journal of Tuberculosis and Diseases of the Chest*, October, 1944) have used carbon dioxide by inhalation as an expectorant in pulmonary tuberculosis for many years and find it most efficient. Its liquefying effect upon mucopus in the bronchial tract is greater than that of any of the commonly used expectorants. Moreover, they say, by its action upon the respiratory centre it causes increased expansion of the chest, a consequent stretching and dilatation of the bronchi and an increase in bronchial peristalsis. They claim that these factors produce a greater amount of expectoration, so that the resulting adequate evacuation of the bronchi ensures comparatively long periods of freedom from cough. The gas is administered in a mixture containing 10% carbon dioxide and 90% oxygen, which the authors state is a "safe and feasible procedure", and is inhaled through a "B.L.B." mask at the rate of four or five litres per minute for from five to fifteen minutes, once, twice and thrice daily. The authors allow patients who do not like the mask to inhale the gas through a glass tube, and find the results satisfactory, notwithstanding admixture of air and dilution of the carbon dioxide.

Effect of Disease of the Mitral Valve on the Electrocardiographic Pattern.

BENJAMIN V. WHITE, RALPH C. PARKER AND ARTHUR M. MASTER (*Archives of Internal Medicine*, August, 1944) evaluate the electrocardiographic changes commonly recognized in disease of the mitral valve with particular reference to such diagnostic value as they may possess. The inquiry was undertaken inasmuch as universally recognized standards of abnormality of the *P* wave and deviation of the axis do not exist. Electrocardiographs were analysed from a group of 100 controls, a group of 100 subjects with functional systolic murmurs, a group of 43 subjects with mitral insufficiency, and a group of 22 patients with mitral stenosis. The criteria considered were the heights of the *P* waves in the standard leads, the width of the widest *P* wave in any lead, the degree of notching of the *P* waves and the leads in which it occurred, and the determination of the direction of the electrical axis. Certain variations of the *P* waves were found to be of statistical significance, and tentative criteria of abnormality were established, a study of which showed them to be of corroborative but not of specific diagnostic value. It was found that a group of subjects with functional systolic murmurs showed no difference in the electrocardiographic patterns from a control group of similar age. Changes in the *P* waves occurred in some subjects with mitral insufficiency, but they were not sufficiently frequent to be of diagnostic importance. The patients with mitral stenosis showed a significant percentage of tall *P* waves (3·0 millimetres or more), broad *P* waves (0·12 second), and *P* waves which were deeply notched in two or more leads. One or more of these changes occurred in 62% of such patients, as compared with 4% of controls. The tall *P* waves in mitral stenosis appeared as a rule in leads II and III, rather than, as is commonly taught, in leads I and II. The angle of the electrical axis was found to be of no diagnostic value.

Primary Atypical Pneumonia.

S. GUNDERSEN (*The New England Journal of Medicine*, November 23, 1944) discusses primary atypical pneumonia of unknown ætiology. He describes

symptoms which might be found in any case of pneumococcal pneumonia, the only distinguishing point being the mildness of the disease. It has been assumed apparently that if pneumococci are not demonstrated in the sputum, the condition is definitely not pneumococcal. The number of sputum examinations is not stated, but the sputum was examined in every case in the author's series. In 82 cases many organisms were found in the cultures, but none of them predominated. Green-producing streptococci, staphylococci, pneumococci, *Micrococcus catarrhalis* and influenza bacilli were the organisms most frequently found. It seems remarkable, in view of the organisms present in the sputum, that the diagnosis of primary atypical pneumonia of unknown ætiology should be attached to this type of pneumonia. Atypical pneumonia and mild or larval pneumonia are very well known entities. Indeed experienced physicians might assert that they see a great many more atypical than typical pneumonias. Surely typical pneumonia is rather a rarity. Osler has referred to mild or larval pneumonia in his "Principles and Practice of Medicine". It would appear to be scientifically wrong to invent and perpetuate a name such as primary atypical pneumonia if indeed the condition is already well known. The proof that the ætiology is unknown rests in this series on one sputum test; and in 82 out of 122 cases staphylococci, pneumococci and other organisms were found in the sputum. The same comments might be made in the diagnosis of virus pneumonia in many cases. This diagnosis is often made in mild pneumococcal pneumonia without any justification. The practitioner should beware of diagnosing primary atypical pneumonia or virus pneumonia unless he is certain that the condition is not due to pneumococci, streptococci or one of the usual causes of pneumonia.

Pneumonia.

E. S. MILLS AND D. MCCALLUM (*The Canadian Medical Association Journal*, December, 1944) report 425 cases of pneumonia in Montreal in the period 1941-1943. These cases included those in which patients were admitted to hospital suffering from pneumonia or developed pneumonia in hospital, but excluded post-operative bronchopneumonia and pneumonia occurring as an agonal event in the course of other diseases. The sputum was examined in all cases. There were 392 cases of pneumococcal pneumonia, and 49% of these were due to pneumococci of types I, II or III. Twenty-seven deaths occurred; they were mainly due to intercurrent disease or to delay in the treatment of patients admitted to hospital with bacteriæmia; 50% died. The results of treatment with sulphadiazine and sulphathiazole were equally good. Six cases of *Staphylococcus aureus* pneumonia were noted. Two patients did not respond to sulphadiazine and were given sulphathiazole with success. One case of Friedländer's pneumonia occurred, and there were 26 cases in which no organism was isolated. In none of these was the pneumonia lobar in distribution, though 24 patients had X-ray evidence of lung disease, mainly in the form of irregular, hazy shadows peripherally or at the hilus. There was, however, no clear-cut clinical picture, and all degrees of severity were observed; nor was there any correlation between the apparent severity of the infection and the radiological findings. It would appear that there is no sharp distinction between cases in which only upper respiratory tract infection is present and those in which lung involvement is obvious. The infecting agent may involve only the upper respiratory passages, or may spread to smaller branches of the respiratory tree with more severe and prolonged fever. In neither of these types is there any radiological evidence of disease. In other cases the infection may involve the lung proper, producing evidence of pneumonia, peribronchial in distribution, with high fever, leucocytosis and prostration, but few physical signs.

Electrocardiography.

G. W. MANNING AND C. B. STEWART (*The Canadian Medical Association Journal*, December, 1944) describe alterations in the *P-R* interval of the electrocardiogram with change of posture. In the case of three air crew trainees the *P-R* interval was 0·20 second with the trainees in the sitting posture, and changed at once to 0·28, 0·24 and 0·24 when the subjects assumed the recumbent position. In a fourth case the *P-R* interval was 0·40 second with the subject in the recumbent posture and 0·20 second when he was in the sitting posture. In no case was there any significant change in the heart rate. No other abnormality was detected. One of these airmen served for two years as a fighter pilot overseas after these facts had been observed. Further electrocardiograms four years later revealed no change. The *P-R* interval could be reduced to normal limits by excessive exercise in the recumbent posture or by the assumption of the upright position. The *P-R* interval was observed to change just as the erect posture was reached, and became prolonged again just as the horizontal posture was reached.

The Transmission of Infective Hepatitis.

W. P. HAVENS *et alii* (*The Lancet*, February 17, 1945) have shown that the virus or "icterogenic agent" of infective hepatitis is in the fæces of the sufferer, and that the disease can be transmitted to others by the administration of the fæcal material in capsules or by spraying it into the nose and pharynx. It is found in such cases that the incubation period has been on the average about twenty-eight days. The authors conclude that "there is justification for suspecting that the intestinal-oral circuit is part at least of the natural route of spreading infective hepatitis".

Achalasia of the Cardia.

C. E. FIELD (*The Lancet*, December 30, 1944) has described the treatment of achalasia of the cardia with octyl nitrite. Amyl nitrite and glyceryl trinitrite have been used with success in this condition. Amyl nitrite appeared to be much more effective than glyceryl trinitrite, but its nauseating effect made its use undesirable for children. Octyl nitrite was less unpleasant to inhale, though its untoward effects were similar to those of the other nitrites. An inhaler designed in America was used for this drug. Five patients were treated by inhalation, one breath causing the cardia to open. One single inhalation at the end of a meal, or a small inhalation in the middle and one at the end of the meal caused the cardia to open. The patients all showed clinical improvement, but no spontaneous relaxation of the cardia has been observed.

Peptic Ulcer.

J. N. MORRIS AND R. M. TITMUSS (*The Lancet*, December 30, 1944) have discussed the epidemiology of peptic ulcer. In ten years before the war there were 43,200 deaths from peptic ulcer in England and Wales. There has been a steady rise in the death rate from this cause and it has been accelerated during the war. In males peptic ulcer has increased both in the old and in the young and middle-aged. During the depression of 1930 to 1935, when unemployment was rife, the mortality of peptic ulcer declined to a marked extent, only to rise again with an increased employment of individuals. Although clinically duodenal ulcer is said to occur much more frequently than gastric ulcer, deaths are higher for gastric ulcer than for duodenal ulcer. There has been no marked rise in the mortality rates for women from either cause. Up to December, 1941, 23,000 men were discharged from the army on account of peptic ulcer. So far as social factors are concerned it is noted that poverty and gastric ulcer are associated in the young, but duodenal ulcer causes more deaths in the well-to-do in later life.

Syphilis and Penicillin.

R. O. F. ROSS, R. B. NELSON, E. M. LOUIE AND H. O. J. COLLIER (*The Lancet*, December 30, 1944) have described the treatment with penicillin of five patients suffering from early syphilis. Patients with well-marked secondary lesions were treated; 30,000 units were injected intramuscularly every three hours for 80 injections, or 30,000 units were given intramuscularly every hour until 40 injections had been given. The immediate response in all cases was excellent, being at least as good as that with arsenicals and bismuth. However, later effects were not so good. In only one case was an unequivocal success achieved at the end of nine months.

Hæmothorax.

T. HOLMES SELLORS (*The Lancet*, February 3, 1945) states that the liquid condition of the blood in most hæmothoraces has been the basis of much speculation and that theories have been advanced on the assumption that clotting has not taken place. However, clotting does occur at ordinary speed, but owing to "the physiomechanics of the chest" (chiefly to the turbulent motion to which the pleural contents are subjected) the clot becomes rapidly defibrinated, so that the gel form of the clot is altered to shreds of fibrin floating about in a pool of liquid blood containing practically no fibrinogen. The author advises early aspiration of hæmothoraces and states that the fear of starting fresh bleeding is a groundless one. In the rare cases in which blood in a hæmothorax becomes clotted thoracotomy is indicated. Breathing exercises are of great value in re-establishing full expansion of the collapsed lung.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

A MEETING of the Melbourne Pædiatric Society was held on March 14, 1945, at the Children's Hospital, Carlton, Melbourne, DR. ERIC PRICE, the President, in the chair.

Prophylactic Treatment of Rheumatism with Sulphanilamide.

DR. G. ANDERSON read a paper entitled "Prophylactic Treatment of Rheumatism with Sulphanilamide" (see page 635).

DR. HENRY SINN paid a tribute to the energy, tact and skill shown by Dr. Anderson in the course of his investigations. Dr. Sinn said that Dr. Anderson was courteous both to the honorary members of the staff of the rheumatic clinic and to the patients themselves. It might be held by some that investigations of this nature were superfluous, as the subject had been exhausted in the American literature. But different conditions were operating in Australia, and a study of the papers already published revealed no unanimity on the subject. In fact, the reverse existed, for there was a wide divergence of opinion, as could be seen by some short extracts which Dr. Sinn said he had taken from a few articles available. Caroline B. Thomas, R. France and Franjo Reichsman, in *The Journal of the American Medical Association* of February 15, 1941, at page 551, published the results of treatment of 55 patients with 18 grains per day of sulphanilamide, and details of 67 control cases; no patient had relapsed in the former group and 15 relapses had occurred in the control group. There were four deaths in the latter group, two from subacute bacterial endocarditis. The only toxic effects experienced were skin eruptions and leucopenia without granulocytopenia. Caroline A. Chandler and Helen B. Tausig, in *The Bulletin of the Johns Hopkins Hospital* for January, 1943, at page 42, reported having given sixteen children sulphanilamide and having withheld the drug from seventeen. There was one doubtful recurrence amongst the sixteen, and five unquestionable recurrences amongst the controls. These patients were studied over a period of two years, and included some who had suffered an acute rheumatic episode within three years and whose condition was quiescent for at least three months. Ann G. Kuttner and Gertrude Reyersbach, in *The Journal of Clinical Investigation* for January, 1943, at page 77, reported having given 108 children prophylactic sulphanilamide treatment and having studied 104 controls. Two of the treated group developed streptococcal throat; one of these patients had a rheumatic relapse. Forty-eight of the controls developed streptococcal throats and twenty-eight suffered from rheumatic fever. Dr. Sinn said that these results were highly gratifying, but that the other side of the picture was revealed by the next two extracts which he had studied. C. R. Messeloff and M. H. Robbins, in *The Journal of Laboratory and Clinical Medicine* for August, 1943, at page 1323, reported having given five patients prophylactic sulphanilamide treatment and compared the results obtained among thirty controls. Three treated children had rheumatic relapses, and two of these died; relapse occurred also in three of the controls, and one death from heart failure took place. These investigators administered a dose of fifteen to twenty grains of the drug per day over a period of eight months. The toxic effects observed were anorexia in one case, reduction of hæmoglobin value in four cases, mild leucopenia in six cases, mild granulocytopenia in two cases and a maculopapular eruption in each of two cases. The authors concluded that the drug had proved of no value in this series. D. D. Stowell and W. H. Button, junior, concluded after their study of a small group of cases that the drug should not be used and could be lethal (*The Journal of the American Medical Association*, December 20, 1941, page 2164). One of their patients died from acute agranulocytosis with secondary infection of the blood stream by hæmolytic *Staphylococcus aureus* and pneumococcus type II. In five out of fourteen cases of another group treated with sulphanilamide, the drug had to be stopped because of toxic reactions.

Dr. Sinn said that this small series of abstracts was sufficient to show that the subject was still unsettled and the field remained open for further investigation. It was worthy of note that Dr. Anderson had a much larger number of cases at his disposal than these American workers. Although it was obvious that studies of this nature were of great value, it was imperative not to over-emphasize their importance, especially to the point of ignoring other considerations in the prevention of rheumatic infections and relapses. Dr. Sinn said that the problem of prevention in this disease would remain sociological rather than therapeutic. Efforts to raise the standard of living, to enhance nutrition and to improve the housing conditions of the poor must not be relaxed because of any results, imaginary or real, obtained by sulphanilamide and its allies. The role played by chronic tonsillar infection and dental sepsis also had to be taken into consideration; and last but not least, it had been shown that salicylate or aspirin administered during the interim between the throat infection and the rheumatic episode had found a place in the preventive treatment of this disease.

Dr. Sinn said that Dr. Anderson had related a number of interesting observations not immediately concerned with the main study. He, too, wished to record an interesting side effect which was brought to his attention. This was the inability of sulphanilamide in the dosage given to prevent the common recurring "colds" or upper respiratory tract infections apart from streptococcal throat. Frequently the mothers replied in answer to the routine *questionnaire* that the child had been very well while taking the tablets, but had another "cold". Optimists who hoped to prevent these infections were apparently doomed to disappointment.

Dr. Sinn went on to say that it had always seemed anomalous to him that the drug might be useful in preventing relapses when most authorities agreed that it was contraindicated during acute attacks. But medicine was full of such anomalies, and only by investigations such as that which Dr. Anderson was carrying out could such questions be resolved. The hospital was fortunate to have men of Dr. Anderson's calibre to help with this important work. He would be missed when he took up his new appointment. The standard set in this work as well as in his other undertakings augured well for his future success. Dr. Sinn said that he hoped Dr. Anderson would not divorce himself from clinical medicine when he took up research work, and he wished him well.

DR. J. W. GRIEVE supported the remarks made by Dr. Sinn. He thought that the investigations should be continued.

DR. ROBERT SOUTHBY offered his congratulations to Dr. Anderson on his excellent paper. Dr. Southby said that if the figures so far obtained could be maintained, sulphanilamide therapy would be a decided advance, especially from the point of view of relieving the bed shortage for these children and overcoming the difficulties of dealing with such cases in the out-patient department. The cooperation of mothers and children was most helpful; this depended largely on the person in charge of the clinic, and the good attendances were attributable in no small measure to Dr. Anderson's conscientiousness. Dr. Southby said that he understood that the reason for the frequent observations of the blood in the early weeks was based on the hypothesis that intolerance to the drug manifested itself in the first six weeks, and if this period was passed safely the likelihood of toxic effects was remote.

DR. GUY SPRINGTHORPE said that he regarded this work as a first-class piece of clinical research of a type rather rare nowadays. He wondered what proportion of the patients in each group had cardiac lesions and if they were equally divided in the two groups. He also wondered whether there had been any increase in the cardiac disease during the course of treatment. Dr. Springthorpe remembered reading the results of research into the question of tonsillectomy in rheumatic patients. As far as he could recall, this procedure had not reduced the incidence of first or subsequent attacks. Dr. Springthorpe said that he thought it was necessary for Dr. Anderson to carry on with his investigations so that these and other questions could be resolved.

DR. H. MCLORINAN also said that he appreciated Dr. Anderson's paper. Dr. McLorinan understood that the chief objections to sulphanilamide therapy were that the organisms might become sulphanilamide-fast and the patient sulphanilamide-sensitive. With the exclusion of such a valuable drug, the chances of recovery from this and other diseases which the patient might contract might be prejudiced. However, the first objection was not really considerable, and the introduction of penicillin as a therapeutic agent considerably reduced the weight of the second objection. Dr. McLorinan thought that these considerations should not deter Dr. Anderson from proceeding with further investigations.

DR. NOREEN NICHOLSON asked Dr. Anderson whether the dosage should be amended in certain cases and especially for adults. In four cases, the treatment was being carried out at the Alfred Hospital. Therapy had just been commenced in one case. Another patient developed rheumatic

manifestations two days after the drug was first administered. In the other two cases, blood examinations revealed little or no elevation of the sulphanilamide level in the blood stream after six weeks' treatment.

DR. MONA BLANCH asked how long sulphanilamide therapy was to be continued.

SQUADRON LEADER D. G. HAMILTON said that it was necessary to compare the sociological background in the two groups. Dr. Anderson had selected patients whose parents were intelligent enough to cooperate. These may have come from better homes. It was necessary to find out whether the home conditions of the control group compared with those of the treated group and to see if the intelligence of the parents was comparable in the two groups.

DR. ERIC PRICE said that he thought the congratulatory remarks made to Dr. Anderson were most deserved. He asked what might be the possible effects of sulphanilamide as a preventive in other streptococcal or toxic diseases. He wondered whether the drug might prove beneficial to patients with the so-called paraarticular manifestations of rheumatism—lesions in the heart, pericardium and central nervous system.

Dr. Anderson, in reply, thanked the various speakers for their remarks. He said that Dr. Sinn had rightly pointed out that the work was experimental and was only one weapon available to deal with rheumatic recurrences. Stowell and Button suspended their investigations because of the incidence of agranulocytosis in one of their patients. To avert this danger, frequent blood investigations were carried out on the patients treated in Dr. Anderson's series. Dodge *et alii*, writing in *The Journal of Pediatrics* of May, 1944, pointed out that all cases of agranulocytosis occurred within the first six weeks of administration of sulphanilamide; not all these patients were rheumatic. In the majority the condition developed between the eighth and fourteenth days. It was apparently an allergic reaction due to sensitivity to the drug. This gave point to the Russian view that to counteract the effect more sulphanilamide should be given. With regard to the prevention of "colds", Dr. Anderson said that the investigations had shown that "colds" and sore throats, except those due to hæmolytic streptococcus group A, were not prevented by sulphanilamide. Indeed, hæmolytic streptococcus group B appeared to flourish in its presence.

Sulphanilamide was first employed to overcome the acute phase of rheumatic fever. But it was found that the disease was made worse by the drug. Dr. Anderson said that it was possible that the destruction of organisms by sulphanilamide might liberate toxins and so aggravate the condition. Professor Rubbo was interested in the theory postulated in America that the manifestations of rheumatic fever were due to sensitivity to a toxin-collagen compound which accumulated in the disease and acted as an antigen. Antibodies were formed and an antigen-antibody reaction occurred.

With regard to Dr. Springthorpe's inquiries about the progress of cardiac lesions under treatment, Dr. Anderson said that the drug was not given to any children without clinical evidence of cardiac involvement (cardiac murmurs). In a few of the cases other murmurs had developed or the murmur had changed in character, but the clinicians in charge of the clinic believed that these changes were due to fibrosis occurring in the valves. In this series, the tonsils were removed from a number of children because they were infected, and not in an attempt to prevent recurrence. It was true, as Dr. Springthorpe had said, that tonsillectomy did not reduce the incidence of the disease or influence relapses.

Dr. Anderson paid a tribute to the ever-ready help and cooperation furnished by Dr. H. L. Stokes with this work. Dr. Anderson regretted that Dr. Stokes was unable to be present at the meeting.

In reply to Dr. Southby, Dr. Anderson said that the regular attendances were in no small measure due to the appointment system which was used during the investigation. The patients were given a card with their name and date and the time at which they were to attend again. Also included on the card were a list of symptoms that they should report to Dr. Anderson. The patients kept the cards and were proud of them. It was hoped in time to have all the rheumatic patients attending by appointment at specified times. As Dr. Southby had remarked, the first six weeks were the dangerous period as far as toxic effects were concerned. Dr. Anderson said that he saw the point of the objections raised by Dr. McLorinan. It was felt that these dangers could be risked, and so far he had had no reason to regret that he had taken the chance. One child did return to the hospital with pneumonia and did indeed respond slowly to sulphanilamide therapy. However, the introduction of penicillin had provided a new weapon to help in such instances. In reply to Dr. Nicholson's question, Dr. Anderson said that the dosage for larger children was three tablets per day. In three or four cases the blood sulphanilamide level was found to be zero or near zero. When these patients were admitted to hospital and observed, the blood level rose without change in the treatment to 1·5 or 2·0 milligrammes *per centum*. It was therefore thought that the children had not been taking the drug, and this fact might account for the low levels recorded for Dr. Nicholson's patients. In reply to Dr. Blanch's inquiry about the length of time for which the patients received treatment, Dr. Anderson said that in America treatment had been pursued for five or six years. The question had not been definitely decided, but possibly it was necessary to give the drug for a number of years. The high incidence of recurrence after the drug had been stopped had been the subject of comment in America. In the treatment of one child at the clinic sulphanilamide was inadvertently suspended, and soon afterwards the patient developed arthritis and *erythema marginatum*.

Dr. Anderson went on to say that Squadron Leader Hamilton had raised the question that the selected patients might have been children of intelligent parents of good sociological background, who would not have had recurrences in any case. As a matter of fact, only twelve of fifteen children were excluded from the treatment because their parents were dull and cooperation was therefore uncertain. On the other hand, it was well to remember that all patients treated had at least one attack. Therefore, Dr. Anderson did not think the good results obtained could be explained in this way. In answer to Dr. Price, Dr. Anderson said that he could not express an opinion on the effect of the drug in para-rheumatic infections. Dr. McLaren had expressed a wish to try this therapy in the prophylaxis of nephritis, but so far it had been impossible to carry out this work. In conclusion, Dr. Anderson again thanked the various speakers for contributing to the discussion and for their commendation.

Obituary.

ROBERT GRAHAM BROWN.

THE death of Dr. Robert Graham Brown, which was announced some weeks ago in these pages, has deprived the medical profession in Australia of one of its most notable practitioners in the field of oto-rhino-laryngology. The value of his work was recognized not only in his own State but far beyond its borders, for many who did not know his work at first hand knew of it through his writings.

Robert Graham Brown was the son of the late John Graham Brown, of Brisbane. He studied medicine at London and took the conjoint qualification L.R.C.P. and M.R.C.S. in 1912. He turned his attention almost at once to ophthalmology and to oto-rhino-laryngology. For a while, however, he acted as full-time demonstrator in anatomy at London Hospital. He then held resident appointments at the same hospital in ophthalmology and in oto-rhino-laryngology. Soon after this war broke out, and he joined up with the Royal Army Medical Corps, serving as ophthalmic specialist with the rank of captain. After demobilization he returned to Australia and joined the late Dr. William Nathaniel Robertson in ear, nose and throat practice at Wickham Terrace, at the same time being appointed Assistant Honorary Ear, Nose and Throat Surgeon at the Children's Hospital, Brisbane. He succeeded W. N. Robertson in the appointment of full surgeon. He held another appointment at the Mater Misericordiæ Public Hospital. For a period he served as surgeon in the ear, nose and throat department of the Brisbane General Hospital. In 1920 he resigned from the staff of the Children's Hospital on the introduction of a regulation precluding the simultaneous holding of more than one honorary appointment. It was not long before he gave up all honorary hospital work.

Robert Graham Brown was always a student. He read widely in medicine and knew the latest views of those who contributed to the literature of ear, nose and throat surgery. He made many journeys to England, Europe and America. In every place he contrived to know the man as well as his work and to keep in touch with many of them after his

return to Australia. In this way he kept himself to an unusual degree abreast of newer developments. Graham Brown was a keen critic and would argue a point at great length. It was characteristic of him that if he thought a certain method not suitable or an opinion incorrect, he would show why he held his own views; his arguments were generally constructive as well as destructive. Because of a good and complete method of keeping records in his practice, he could at a moment's notice produce histories of patients who had suffered from this or that disability, and the notes were always complete with photographs and reports on specimens, and quite often the mounted specimen could be produced on demand. He became a Fellow of the Royal Australasian College of Surgeons and was always at hand when medical congresses were taking place. He took an active part in discussions and contributed many papers himself. At the third session of the Australasian Medical Congress (British Medical Association) held at Sydney in 1929 he was president of the Section of Oto-Rhino-Laryngology, and at the centenary meeting of the British Medical Association at London in 1932, a vice-president of the Section of Oto-Rhino-Laryngology. Robert Graham Brown made his first contribution to this journal in 1917, and his subsequent contributions to this and other journals were important. As a reviewer of books he was of much assistance to the journal for a period of many years.

Robert Graham Brown was an unusual man in many ways. He combined scientific ardour with a love of art and of nature. He took a lively interest in his garden, was a good landscape gardener and had almost a professional pride in his shrubs and ferns, designing and making his own fern houses and fashioning cement pillars for fountains and other ornamental devices. Having visited most of the famous art galleries of Europe, he had a wide knowledge and understanding of art and its treasures. He had at his home an interesting collection of pictures that gave much pleasure to him and to his visitors. He was a student of architecture and knew and loved many of the works of the masters in music. Saint John's Cathedral, Brisbane, has been enriched by several of his gifts—a sculptured plaque, stained glass windows, and a triptych—in memory of his parents and one of his friends. For all this he was often a lonely man. He lived almost in several compartments. It was difficult for some of his colleagues to know him. He had a manner that often kept men at a distance; he could be brusque, and latterly, when he was tired and when his health was not good, his judgements were occasionally hasty and even harsh. To his intimate friends it seemed sometimes that there was, behind a seeming turbulence of spirit, a shy reserve, and that this was the manifestation of a sensitive nature that had been hurt in the vicissitudes of a full and busy life. His sudden death, which occurred while he was working in his garden, was what in all probability he would have wished. As already mentioned, he enriched the world of medical science, but he also left behind him a wealth of esteem and affection among people in every walk of life.

DR. G. C. SCANTLEBURY writes: By the death of Robert Graham Brown, Australian laryngologists have lost a colleague of whom they were very proud and whose personality was always stimulating. An indefatigable worker whilst in his own town of Brisbane, he was constantly travelling to all parts of Britain, the Continent and the United States of America wherever he thought some interesting work was being done. At home again in Australia, he spared himself no effort in taking a leading part in any congress. Subjects such as the surgical treatment of carcinoma of the larynx, the radiological investigation and surgery of the nasal sinuses and plastic procedures generally were discussed by him most thoroughly in freely illustrated addresses and papers. These were frequently followed by actual operations not only in his own theatres. Local anæsthesia with its premedication was forever one of his special pleas. At the Royal Melbourne Hospital on one occasion he demonstrated his local anæsthesia technique in radical antrostomy which had a big effect on the subsequent operation there. A great exponent of local anæsthesia in tonsillectomy, he took his own instruments to America (he ruefully told the writer that he had to pay duty on these) to demonstrate his method to Dr. Fowler in New York. A colleague of his in Brisbane once remarked: "One great advantage of Brown being in Brisbane is that one is kept constantly on one's toes." Wherever he was, one could always count on an argument, but such was ever constructive.

Apart from medicine and surgery, he had many and varied interests. Paintings and drawings always interested him and he was quite an authority on landscape gardening. His gift of a fully equipped ambulance to the services early in the war was one of many acts of generosity, and his kindness to his patients was proverbial. I speak for the whole profession when I deeply deplore his loss and extend to his widow our sincere sympathy.

DR. A. B. K. WATKINS writes: The passing of Dr. Robert Graham Brown leaves a gap in the ranks of oto-rhino-laryngologists which it will prove hard to fill.

When I met him first in 1924, we were attracted by having been trained at the same medical school at the London Hospital. Gradually I got to know and like him well and to respect his many great qualities. He was a remarkable man from many aspects. His habit of frequently leaving his practice to visit clinics abroad made him probably both the most travelled Australian ear, nose and throat surgeon and the best informed on work likely to be later published. He knew personally practically all of his more famous contemporaries.

This habit of travelling detracted little from the size of his practice, for when in Brisbane he worked with overpowering energy and forcefulness. I have seen him perform twenty operations in an afternoon session, and he told me he often operated on over 400 cases monthly. He did so with a speed, deftness and certainty of touch equalled by only a few general surgeons and by no other oto-rhino-laryngologist whose technique I have watched in this or any other country. The number of operation cases he undertook was such that he could afford no loss of time. This no doubt accounted for any intolerance he may have displayed at any but first-class assistance from the nursing or medical teams associated with him.

His interest in surgery overflowed his immediate speciality and he did much skilful work on the thyreoid, glands of neck, lips and palate. He was one of the earlier pioneers of local anæsthesia in Australia, and his feats in this field were rendered remarkable by the extent of anæsthesia he could produce with the minimum of anæsthetic solution and of premedicant used.

He made many contributions to medical literature, one of the best known being a long article in *The Journal of*

Laryngology and Otology on radiological appearances in sinusitis.

Once away from his surgery or from the hospital, his professional worries dropped from him as a cloak and he became a delightful host, showed an interest in oil paintings, furniture, gardening, and many subjects one would not have expected him to have had the opportunity to study. He displayed many strong characteristics, most of which, such as loyalty to his friends and his untiring energy in doing things for anyone he liked, impressed me strongly and drew me to him. His was not the nature which would have tolerated well a gradual enfeeblement of his faculties, and no doubt his sudden passing before this stage was reached is what he would have wished had his been the choice.

DR. VAL MCDOWALL writes: With the passing of Dr. Robert Graham Brown the medical profession in Australia has lost a surgeon whose reputation for efficiency and originality, especially in the sphere of diseases of the ear, nose and throat, extended to both England and the United States of America.

He commenced his university education in the Arts School in Sydney, but after a short time went to London, and began the study of medicine at the London Hospital.

In his early years he came under the influence of the late Dr. Hawkes, of this city, and it was mainly his influence that led Dr. Graham Brown, on graduating, to begin the study of diseases of the eye, ear, nose and throat. He worked under many distinguished men, and at one time was demonstrator of anatomy at the London Hospital Medical College.

At the outbreak of the first World War he immediately enlisted with the British Expeditionary Forces, and took part in the famous retreat from Mons. On his return to Brisbane he joined the late Dr. W. N. Robertson and practised in the diseases of the eye, ear, nose and throat. Later the partnership was dissolved and Dr. Graham Brown commenced practice on his own account in Preston House (now the Equitable Life Building), Queen Street, Brisbane, and confined his attention to diseases of the ear, nose and throat. His success was rapid and he soon reached the top of his profession.

At different times he served as honorary ear, nose and throat surgeon on the staffs of the Brisbane General Hospital, the Hospital for Sick Children, and the Mater Public Hospital. The greater part of his professional life, however, was devoted wholly to private practice, where his investigation of his cases was such that he said (and I believe with truth) that he gained as much as in a public hospital practice. His keen interest in his cases was exceptional. No case however difficult but was given a chance, and in some instances these grave and frequently hopeless cases were successful.

His investigations of his cases was very thorough, and his outspoken criticisms sometimes made him enemies amongst the lesser fry of his profession. I have had many a heated argument with him; after an interesting operation he would invariably bring along the specimen and discuss the case to our mutual advantage.

In order to keep himself abreast of the latest developments in his particular speciality he made numerous visits abroad, and contacted other well-known surgeons. His equipment in instruments and books was of the best, and he lavished much time and money in his efforts to get perfection in his instruments. He wrote many original articles in which he carefully and thoroughly worked out his own ideas; probably the best known of these articles were under the headings "Skiagrams in the Diseases of the Maxillary Sinus" and "Considerations in the Diagnosis and Operative Treatment of Cancer of the Larynx". His addresses and demonstrations at various meetings of medical men, and in courses of post-graduate instruction, were models from which all instructors might learn to their advantage.

In 1932 he had his greatest achievement when he was invited as vice-president of the Ear, Nose and Throat Section to the centenary meeting of the British Medical Association in London. He took an active part in the Royal Australasian College of Surgeons, of which he became a Fellow in 1928.

At the time of his death he was engaged in several investigations, in which he had promise of some success, especially in otosclerosis, the problems of water balance, asthma, *et cetera*.

To Robert Graham Brown's generosity at least two members of the medical profession owe the means of completing their courses. This was a matter seldom mentioned by him, but about which I first heard accidentally from one of the doctors concerned. He also gave liberally to war charities, one of his recent gifts being that of an ambulance to the Red Cross Society. He was generous to his friends, and many were the gifts of paintings, engravings, articles of furniture and plants received by them in token of his friendship.

He was a loyal subject of the Crown and an imperialist. He had a great admiration for the present British Prime Minister, Mr. Winston Churchill.

He was a deeply religious man, though he seldom attended church. In Saint John's Cathedral, Brisbane, are several fine memorials by him perpetuating the memory of his father and mother and his friend from boyhood, the late Mr. Lange Powell, to whom he owed much for his interest in matters of art. To commemorate the work of Archbishop Wand in the Diocese of Brisbane, Graham Brown shortly before his death installed a statue of Saint Martin in a niche on an outside wall of Saint Martin's Hospital, where much of his surgical work was done.

He was intensely loyal to his friends, and nothing could ever change his friendship. His death was sudden and unexpected at the relatively early age of fifty-eight years, while working in his garden, where he was wont to spend his leisure hours, and into which he put the same vigour, care and intense interest as he did in his professional work.

He is survived by his sister, Mrs. A. G. Meyer, of Palm Beach, Sydney, and his brother, Dr. Leonard Graham Brown, of London, also a well-known ear, nose and throat surgeon, and an ex-Rhodes scholar.

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"The Relation of Diphtheria and Diphtheria Carriers and Mastoid Disease to Tonsils and Adenoids", *The Journal of Laryngology and Otology*, Volume XLVII, Number 10, 1932.

"The Treatment of Maxillary Sinusitis (contributed by request), THE MEDICAL JOURNAL OF AUSTRALIA, May 12, 1934.

"The Focal Factor in Rheumatism", THE MEDICAL JOURNAL OF AUSTRALIA, July 20, 1935.

"Control of Bleeding in Nose and Throat Operations", THE MEDICAL JOURNAL OF AUSTRALIA, March 14, 1936.

"Hare-Lip: Vilray P. Blair's Modification of Mirault's Operation", THE MEDICAL JOURNAL OF AUSTRALIA, April 11, 1936.

"The Radical Operative Treatment of the Posterior Group of the Upper Nasal Accessory Sinuses", *The Australian and New Zealand Journal of Surgery*, October, 1939, Volume IX, Number 2.

ISAAC JUDAH SILVERMAN.

WE regret to announce the death of Dr. Isaac Judah Silverman, which occurred at Georgetown, District of Columbia, United States of America, on August 6, 1944.

A memoir in *Annals of Internal Medicine*, October, 1944, states that he was fifty-six years of age and graduated from the University of Melbourne in 1911. He practised medicine in Australia, New Zealand, Ontario and Missouri before settling finally in Washington. He was at the time of his death Clinical Professor of Psychiatry and Assistant Chief Psychiatrist at the Gallinger Municipal Hospital, Washington.

WOLFE SOLOMON BROWN.

WE regret to announce the death of Dr. Wolfe Solomon Brown, which occurred on June 9, 1945, at Hunter's Hill, New South Wales.

Notice.

THE inaugural meeting of the Australian Association of Physical Medicine (British Medical Association) will be held in the William H. Crago Council Chamber, British Medical Association House, 135, Macquarie Street, Sydney, on Friday, July 20, 1945, at 8 o'clock p.m. All members of the British Medical Association interested in physical medicine are invited to attend. Further information may be obtained from Dr. B. G. Wade, 135, Macquarie Street, Sydney, who is acting as temporary secretary.

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Kerkenezov, Nicholas, M.B., 1943 (Univ. Sydney), Pennant Hills Road, Carlingford, New South Wales.

Moore, David Robert, Provisional Registration, 1945 (Univ. Sydney), Balmain Hospital, Booth Street, Balmain, New South Wales.

Freeman, Zelman Samuel, M.B., B.S., 1944 (Univ. Sydney), Sydney Hospital, Sydney.

Naval, Military and Air Force.

ACCORDING to the casualty list received on June 11, 1945, Captain N. R. Godby, A.A.M.C., East St. Kilda, Victoria, who was previously reported to be a prisoner of war, is now reported as "not prisoner of war".

Books Received.

"Handbook of Practical Bacteriology: A Guide to Bacteriological Laboratory Work", by T. J. Mackie, C.B.E., M.D., D.P.H., and J. E. McCartney, M.D., D.Sc.; Seventh Edition; 1945. Edinburgh: E. and S. Livingstone Limited. 7¼" x 5¼", pp. 728. Price: 17s. 6d. net.

"Bone-Grafting in the Treatment of Fractures", by J. R. Armstrong, M.D., M.Ch., F.R.C.S., with a Foreword by R. Watson-Jones, B.Sc., M.Ch.Orth., F.R.C.S.; 1945. Edinburgh: E. and S. Livingstone Limited. 9¾" x 7½", pp. 188, with 204 illustrations. Price: 25s. net.

"The 1944 Year Book of Dermatology and Syphilology", edited by Marion B. Sulzberger, M.D., assisted by Rudolf L. Baer, M.D.; 1945. Chicago: The Year Book Publishers, Incorporated. 7¼" x 5¼", pp. 544, with 75 illustrations.

Diary for the Month.

JUNE 26.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 27.—Victorian Branch, B.M.A.: Council Meeting.
JUNE 28.—South Australian Branch, B.M.A.: Annual General Meeting.
JUNE 28.—New South Wales Branch, B.M.A.: Branch Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All Public Health Department appointments.

Editorial Notices.

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